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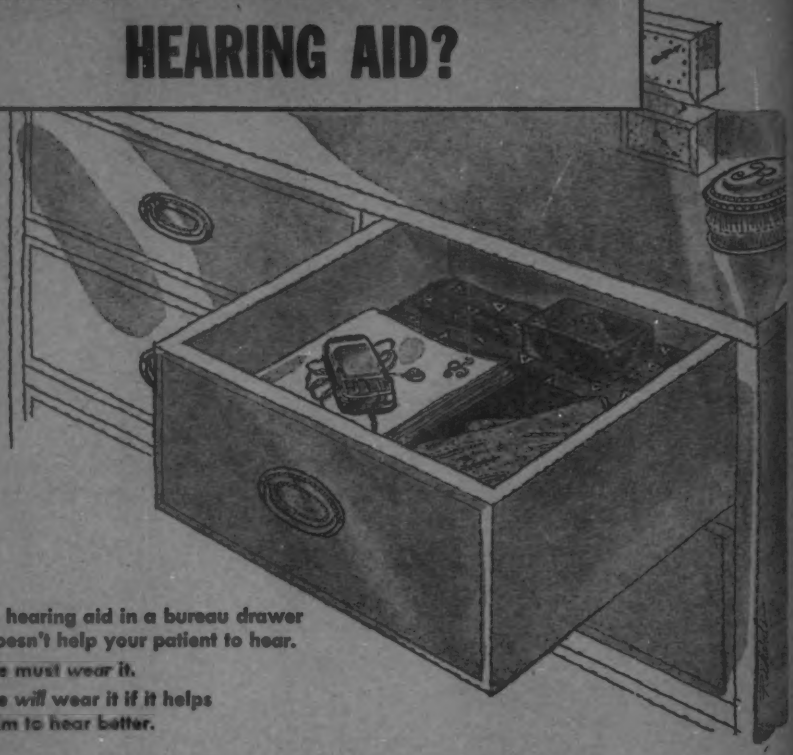

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THE COMBINED USE OF ANTIBIOTIC INSUFFLATION AND VASODILATOR THERAPY IN OZENA.*†

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INTRODUCTION.

Since ozena was first described by Fraenkel[1] in 1876, as a distinct clinical entity, numerous attempts have been made to establish a known etiology as a rational basis for treatment. Failure to solve the problem thus far has led to the growing opinion that the disease is not due to any one single cause but very likely is the product of a combination of factors.[2,3] In view of this trend, the significant though limited results obtained with recent independently formulated methods of streptomycin[3-5] and vasodilator[6-14] therapy suggested a combined approach for clinical investigation. In 1951, I reported[15] a new method for the treatment of ozena by combining the local use of aqueous streptomycin and other antibiotics with vasodilators in oral form. Histamine, priscoline,[16-17] and niacin were employed to stimulate more profound local effects on the vascular and neurotrophic changes through a potential increase in the peripheral circulation of the nose by their combined parasympathetic and sympatholytic actions. The preliminary results suggested that properly selected antibiotics and the multiple use of vasodilators could effectively eliminate odor and crusts and equally tend to promote a reversal of the regressive changes in this disease.

A diagnosis of ozena as differentiated from the secondary form of atrophic rhinitis was based on the following criteria: odor, green crust formation, atrophy and gram negative or-

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† From the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary.

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ganisms consisting of either Friedländer's bacillus, B. coli, B. pyocyaneus or B. proteus.

The purpose of this paper is to present a modification of the earlier use of aqueous antibiotics to the more recent method of insufflation with micronized streptomycin, Terramycin, and chloramphenicol (chloromycetin). The data, results, and follow-up studies in a total series of 35 cases observed over a period of three years are reported including three case reports with X-ray, and microscopic evidence of turbinal growth and regeneration of respiratory epithelium.

LITERATURE.

Etiology: In 1896, when K. ozena was isolated by Abel, it was believed that a bacterial origin of the disease had been discovered.[18] Later investigators found no consistency in the bacterial flora, except possibly for the close morphologically related K. pneumoniae (Friedländer's bacillus)[2,20,22] which proved to be nontransmissible.[19] Further evidence that klebsiella are principally secondary invaders has been suggested.[24a,4,5,27] More recently C. diphtheriae (mitis type) was offered as the primary cause.[42]

Pathogenesis: Pollock (1931) believed that either primary[23] or secondary[24] sphenopalatine ganglion degeneration was induced by preceding sinusitis, resulting in atrophic changes of the nasal mucosa and finally ozena. Heredity, abnormal structural patency, scarlet fever, and diphtheria were mentioned as predisposing and exciting factors. Ballenger[24a] observed, however, that the true cause of ganglion atrophy was still unexplained. Endocrine deficiency and the lack of hormonal secretion was advanced as a principal cause of atrophy by Dorochenko[26] (1937), Mortimer et al[27] (1937), Blaisdell[25] (1938), and Ruskin [28,28a] (1942). Other factors considered by Taptas[29] were toxic destruction of nasal mucosa either primary or secondary to a slow progressive diphtheria of low virulence, and saphrophytic agents resulting from the stagnation of secretion.

Histopathology: No agreement exists as to the uniformity of the particular epithelial and vascular changes inasmuch as adequate histological studies of the so-called normal tur-

binates are lacking. The common pathological findings[2,3,10,30,30a] may be summarized as follows: *a,* Epithelium: metaplasia from ciliated columnar to stratified squamous, with the respiratory mucous membrane still retained in a few protected areas: *b,* Basement membrane: normal to thickened or complete disappearance, *c,* Tunica propria: lymphocytic and plasmocytic infiltration especially concentrated around the mucous glands: minimal to advanced perivascular fibrosis; occasional Russell bodies; deeper vessels thickened, hyalinized and occasionally obliterated; variable fibrosis to atrophy of mucous glands, *d,* Bone: progressive lacunar resorption of the turbinates.

Hormone Therapy: In 1937 and 1938, the local use of estrogenic hormones was reported by Dorochenko,[26] Mortimer, et al,[27] Blaisdell,[25] and later others[31,32] with limited results. Biopsy studies[30] failed to reveal definite changes in a single direction.

Antibiotic Therapy: The chemical effectiveness of streptomycin with the Friedländer organism in mice was first successfully reported by Heilman[33] (1945). Clinical studies by Thornell[3,4] and Simonton[5] later showed a disappearance of odor and klebsiella organisms although a less appreciable effect on crusting. Atrophy remained uneffected. Jannoulis[34] reported only a disappearance of odor with combined penicillin and streptomycin.

Vasodilator Therapy: Various investigators reported encouraging results with histamine ionization[10] and acetylcholine[24,24b,9] attributable to an increased blood supply to the nasal mucosa. Others[12,13] claimed equally good results with vitamin PP and nicotinic acid[14] on the assumption that ozena was a dysvitaminosis rather than a vasomotor disorder. Procaine block of the stellate ganglion[35] was also reported to diminish headache and fetor, with increased turbinal vasodilation, as further evidence of a vasomotor neurotrophic disturbance.

Surgical Therapy: More recently various surgical procedures for narrowing the bony nasal vault with osteotomy,[30]

submucosal implants of plastic[39,40] or bone grafts[43] have been favorably reported. Placental tissue implants showed poor results.[37]

CLINICAL MATERIALS

Ten of 35 patients selected for study were follow-up cases from the original group of 13 first reported.[15] The remaining 25 patients have since been newly added. In the total series, six were men and 29 were women ranging in age from 10 to 73 years, with a duration of symptoms from five to 50 years. Eight patients were treated previously in the outpatient department for a period of about 15 years. Nineteen new patients were referred by the clinic and eight were treated privately since the beginning of this work. Estrogenic nasal sprays, (Estrone 1-10,000 to 1-20,000) had been previously used by eight patients over a period of 10 years and in eight cases sinus surgery was followed by estrogen nasal therapy. Serum tests in all cases were negative.

The clinical findings showed 20 patients with marked odor, slight in two and absent in 13. Although no odor could be detected objectively in eight of the 13 patients on forced expiration, it was nevertheless noticeable on the crusts removed by cotton applicator. Crust formation was generally distributed over the turbinates, septum, nasopharynx and posterior surface of the soft palate. It was marked in 27 cases, moderate to slight in seven, and absent in one. Advanced atrophy (3+ to 4+) was seen in 26 patients, less than (1+ to 2+) in seven, and no gross atrophy in two. Sixteen patients gave a history of complete anosmia, seven partial and 12 no loss of the sense of smell. Nasal blockage and discharge were more or less present. No correlation was observed between the degree of odor, crusting and atrophy.

The bacteriological findings obtained from 35 individual cultures showed a variety of gram negative and gram positive organisms with B. Friedländer's (45%), H. staphylococcus aureus (37%), B. coli (37%), B. proteus (20%) and pneumococcus (11%).

X-rays of the sinuses were reported in 50 per cent of the patients as showing inactive pathological changes. The find-

ings were variable consisting of slight to markedly thickened membrane, a few with added decalcification of the ethmoids and some with either uni- or bilateral sclerotic bone changes. One patient revealed cystic membrane of the antrum and one, three small ivory transplants in the septum. Only 28 per cent indicated active suppuration and thickened membranes. Of this group, one-half showed pus in the antrum, one in the frontal sinus with sclerosed bone and four retained secretion in the ethmoids and antrum with marked bone sclerosis in one. The remaining 22 per cent were negative. Marked to moderate atrophy of the middle and inferior turbinates was demonstrated in all of the cases.

BIOPSY STUDIES.

Biopsies were obtained from the under surface of the inferior turbinate in its approximate middle third section including the lateral and medial aspects. The epithelial changes were found to be variable ranging from columnar to transitional and to stratified squamous epithelium. Cilia and goblet cells were absent except for the occasional suggestion of the former in protected areas on the lateral surface. The apparent lack of vascularity noted clinically was at variance with the histological findings for in some of the more advanced cases of atrophy, larger blood vessels were identified in the deeper portions of the tunica propria despite the marked fibrosis and relatively scant number of capillaries in the subepithelial region. No definite correlation was observed between the microscopic picture and the gross clinical findings.

Comment: Mueller[41] points out that a diagnosis of ozena cannot as a rule be made on the basis of a small biopsy alone without due regard to the clinical picture, as for example in the case of less advanced atrophy (1+ to 2+), where perivascular infiltration and fibrosis may well be within normal limits providing the specimen included the deepest layers down to the periosteum of the turbinate. He believes that all so-called "normal" nasal mucous membrane shows chronic inflammation to a greater or lesser degree, *i.e.*, infiltration with lymphocytes and plasmocytes.

METHOD.

Cultures were taken before and during treatment at approximately monthly intervals. Antibiotic therapy was carried out with the insufflation of either Streptomycin (calcium chloride complex*), Terramycin hydrochloride,† or Chloramphenicol (Chloromycetin‡) in an effort to obtain concentrated effective local administration and dosage. Streptomycin† and Terramycin† were micronized (10 microns, average size) to facilitate their better distribution throughout the nose and nasopharynx. The micronized antibiotics were mixed with sodium lauryl sulfate, a surfactant, diluted with beta lactose powder and dispensed in capsules of two strengths§: the weaker, containing 25 mgs. of antibiotic, 2 mgs. of sodium lauryl sulfate and 70.5 mgs. of beta lactose; the stronger, 50 mgs. of the antibiotic, 2 mgs. of sodium lauryl sulfate and 33 mgs. of beta lactose. These were prescribed in an average daily dosage ranging from 100 mgs. to 200 mgs. Instructions were given to insert the capsule (size No. 5) in the detachable tip of a De Vilbiss No. 119 powder blower either with the extreme ends of the capsule snipped off, or its contents emptied into the same receptacle. The patient was directed to inhale the powder during insufflation. Stinging sensations and lacrimation were at times encountered. If these side effects were not tolerated, aqueous antibiotics were prescribed instead, using bacitracin (1500-2000 u/cc.) or Streptomycin (25,000 u/cc.)§§ or both.

The use of a particular antibiotic was indicated by the predominant flora and clinical response. With mixed organisms, either streptomycin or one of the broad spectrum antibiotics was employed. When the gram negative organisms became inhibited, further antibiotic therapy was continued with either Terramycin or bacitracin depending upon the culture report, the degree of crusting, and discharge. Gantrisin was prescribed for B. proteus organisms in 25 per cent to 30 per cent

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* Supplied by Merck & Co., Inc., Rahway, New Jersey.
† Supplied by Chas. Pfizer & Co., Inc., Brooklyn 6, New York.
‡ Chloramphenicol (Chloromycetin) was not available in micronized form for investigation until after this paper was written. Further studies are now in progress with the new material supplied by Parke, Davis & Co.
§ Prepared at the Massachusetts College of Pharmacy under the supervision of Dr. Leslie M. Ohmart, Professor of Pharmacology.
§§ Streptomycin 1 mg. is equivalent to 1000 μ.

aqueous concentrations locally and orally in tablet form (total daily dose 4-6 grams). Sensitivity tests were conducted with concentrations of antibiotics as high as 10 mgs. per cc. of culture fluid. (The maximum level required for systemic concentrations ranges from 0.6 to 256 micrograms per cc. of culture fluid).[18]

Vasodilator therapy was based largely upon the combined use of Priscoline and niacin. Priscoline,*[16,17] was prescribed orally in dosage of 50 mgs. three to four times a day after meals. Patients were informed of the various side reactions to expect such as flushing of the face and upper trunk, tingling of scalp, chilliness or warmth of the extremities, increased palpitation, muscle twitchings, general aches and pains in the extremities, epigastric discomfort or abdominal cramps with increased peristalsis. Blood pressure was periodically measured. If signs of postural hypotension such as a fall in blood pressure, low back pains or general weakness appeared, the drug was temporarily discontinued. It was not employed in suspected cases of gastric ulcer or cardiovascular disease. Niacin was prescribed in doses of 50 mgs. from four to eight tablets daily after meals. No side effects were encountered with this drug.

In the management of nasal hygiene, all patients were instructed to discontinue nasal irrigations. Lamb's wool (sterile) in the form of small plugs loosely inserted in each nostril with sufficient room left for nasal breathing was prescribed several times daily for one-half hour intervals to enhance nasal moisture. Caroid (papaine)† was employed in either powder or liquid form, as a modifying solvent of viscid nasal secretions. The optimum concentration was a six per cent (6%) mixture with a pH of 5.4 in a beta lactose powder base. The powder form was administered through the detachable No. 119 De Vilbiss tip, insufflating one-quarter scoopful first upwards and repeated downwards for each nostril. Beta lactose was chosen to promote a lower pH of the nasal secretion for the inhibition of bacterial activity.[38] The liquid form, five per cent (5%) with a pH of 5.0+ was prepared as fol-

* Supplied by Ciba Pharmaceutical Products, Inc., Summit, New Jersey.
† Supplied by American Ferment Co., Inc., 1450 Broadway, New York, 18, N. Y.

lows: papaine 5 per cent, beta lactose 6 per cent, glycerine 5 per cent, alcohol, 5 per cent, in 100 cc. distilled water and applied either as nose drops in the Proetz position or with an atomizer.

A mimeographed outline of the daily treatment, given to each patient, is shown as follows:

TREATMENT SCHEDULE.

DRUGS	Morning	Afternoon	Evening	Bedtime***
Caroid Powder*				
Liquid				
Lamb's Wool (½ hr.)				
CAPSULES				
Antibiotics (Capsules)**				
(Aqueous)				
Lamb's Wool (½ hr.)				
TABLETS (after meals)				
Priscoline				
Niacin				
VITAMINS				

* Occasionally produces sneezing and blockage.
** Used with powder blower (De Vilbiss No. 119).
*** After 1 glass milk with crackers or toast.

Patients were instructed to omit treatment on their scheduled appointments so as to evaluate the clinical progress. Anosmia was appraised both from the subjective history and a blindfold test of various odors such as coffee, lemon, cooking spices, perfumes and alcohol. Metabolic, nutritional, and hematological problems were followed in consultation with the medical department of the Massachusetts General Hospital.

RESULTS.

In the combined total series of 35 patients, 17 (48.6%) showed striking improvement, 13 (37.1%) significant, three (8.6%) slight, and two (5.7%) no improvement. Patients classified as strikingly improved had a complete disappearance of odor, crusting and organisms of the klebsiella group. Local examination showed the return of normal sized turbinates with an increase in secretion, circulatory filling, and the sense of smell to a greater or lesser degree. In the group with significant improvement, there was a complete disappearance of odor, a marked decrease in crusts, some increase in

the moisture and turbinal filling, and complete inhibition of the klebsiella organisms. There was no return of the sense of smell. Those considered slightly improved showed complete or partial disappearance of odor, slight reduction, in the crusts, and no change in the atrophy. The klebsiella organisms were inhibited to a variable degree.

More than 50 per cent of the strikingly improved patients were treated with streptomycin, but of these nearly one-half later required either the additional use of Chloramphenicol or Terramycin. In the remainder of the group, Terramycin was administered more often than Chloramphenicol. Those showing significant improvement needed more prolonged antibiotic therapy. The majority of these cases were benefited by Terramycin. In both the striking and significantly improved groups, aqueous antibiotics and Gantrisin were also used locally and orally depending upon the bacterial flora and clinical response obtained. Friedländer's bacillus was completely controlled, while B. coli was inhibited by 50 per cent. The two most resistant strains were H. staphylococcus aureus (37%) and B. proteus (14%), the latter showing less frequency after treatment.

In the follow-up of the 17 strikingly improved cases, seven have been able to maintain their continuous recovery without antibiotics or local treatment over a period of four months to approximately three years. Three of the seven patients with fully recovered turbinates have shown no recurrence of their atrophy after going without vasodilators from five months to nearly two years. The remaining 12 patients have continued in varying degree with vasodilator therapy and the hygienic use of powdered Caroid of 6 per cent and lamb's wool. No harmful side effects have been noted from the prolonged use of the antibiotics and vasodilators. Of the 12 patients in the strikingly improved group who showed marked atrophy (3+ to 4+) prior to vasodilator therapy, seven revealed turbinates that looked relatively normal in size and appearance after two and one-half months to one year of treatment. Six of the seven with less advanced atrophy (1+ to 2+) showed normal looking turbinates after one to six months of treatment. In 77 per cent of the 13 patients with

normal looking turbinates, X-rays taken confirmed the clinical evidence of turbinal recovery (see Figs. 1, 1a). Although turbinal growth was not fully shown by X-ray in the remaining cases, it was clinically evident that increased turbinal filling preceded demonstrable X-ray changes.

Fig. 1. A. N. Female. Age 31. Before treatment. Marked Turbinal atrophy. Sinuses negative. Case 13.

Priscoline and niacin in combination appeared to be the vasodilator drugs of choice for promoting an increase in the circulatory filling and secretory functions of the atrophic turbinate. Only one out of five in the early group of 10 cases responded to the addition of histamine, while three out of

the remaining five were strikingly improved without its use. In the newly added group of 25 patients, only three were given histamine combined with Priscoline and niacin. Two (5%) patients developed histamine sensitivity reactions with gastro-intestinal, skin and asthmatic symptoms after several

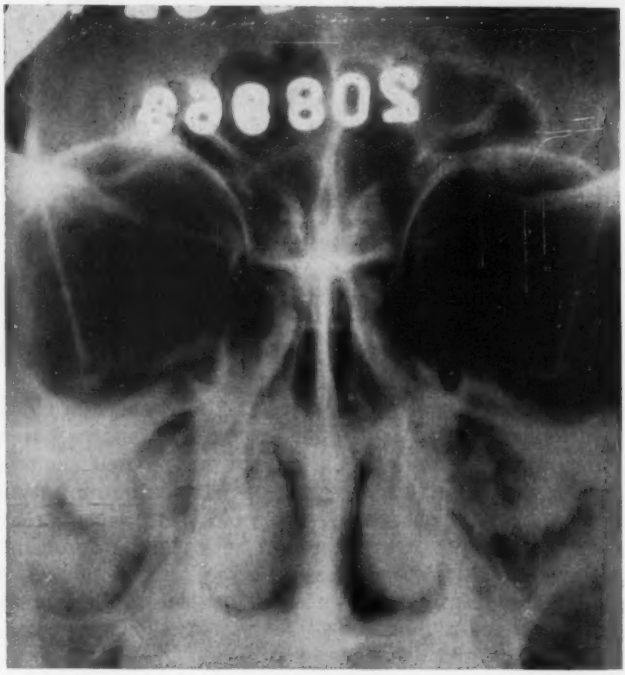

Fig. 1A. After 27 months treatment. Turbinates restored to normal appearance.

weeks of treatment necessitating its elimination. Priscoline intolerance was seen to develop in eight (22.85%) of the 35 cases, although later attempts to resume the drug in some of these patients showed an increased tolerance. The determination as to how long vasodilator therapy should be maintained for its maximum effectiveness remains to be further evaluated.

Of the nine patients with complete anosmia, four showed an appreciable return of their sense of smell and two merely a slight return.

Sensitivity tests revealed gram positive flora to be most responsive to Terramycin and bacitracin. B. Friedländer's, B. coli, and H. influenza were equally responsive to streptomycin, Terramycin and Chloramphenicol. B. proteus and B. pyocyaneus were preponderantly insensitive to the antibiotics. Sensitivity tests as a guide to local specific therapy, however, were generally found to be less accurate and reliable than the actual clinical response obtained.

FOLLOW-UP OF EARLIER CASES WITH AQUEOUS ANTIBIOTICS.

Ten of the 13 patients comprising the earlier series[15] of cases treated with aqueous antibiotics were followed for two to 13 months after the antibiotics were omitted. Vasodilator therapy along with the local use of Caroid and lamb's wool was continued. Two out of four patients in this group maintained their earlier striking improvement for three and 13 months respectively, while the remaining two showed some regression after two and one-half and six months. In the significantly improved cases, three out of four held their gain for two and one-half to three months. The remaining patient returned after an absence of two years with a complete recurrence of symptoms. Of the two with slight improvement, one showed a complete regression after five months. On the basis of these results, the use of insufflation therapy was investigated for a more effective local method of antibiotic administration and dosage.

FOLLOW-UP OF EARLIER CASES WITH ANTIBIOTIC INSUFFLATION.

Eight of the 10 patients were subsequently treated with streptomycin, Terramycin and Chloramphenicol for an additional period of two and one-fourth to 13 months and followed for six to 26 months after the antibiotics were omitted. Vasodilators were continued. Three recovered their earlier striking improvement and have held this result from nine months to two years after antibiotic insufflation was omitted. Two of these, were treated with Terramycin and one with Chloram-

phenicol. The three patients who maintained significant improvement, while making no further clinical progress showed little regression during the six months to more than one year after insufflation was terminated. One patient had been treated with Terramycin, one with Terramycin and later streptomycin, and finally one with all three, of which Terramycin was the most satifactory. The two patients with slight improvement showed no change despite prolonged antibiotic therapy from several months to approximately two years. One was treated with Terramycin and Streptomycin combined, the other with Terramycin followed later by Streptomycin. In the latter patient, vasodilator therapy has been continued for as long as 33 months. This was the only patient whose cultures continued to show Friedländer's bacillus. No correlation was found between the cultural changes in the bacterial flora and the clinical results except for the consistent inhibition of Friedländer's bacillus in nearly all cases.

FOLLOW-UP OF LATER CASES WITH ANTIBIOTIC INSUFFLATION.

Insufflation therapy in the group of the 25 newly added patients was given at various intervals from one week to 15½ months. Combined treatment with vasodilators was simultaneously carried on over a period of 24 months. Thirteen of the 25 were strikingly improved. Ten showed significant, one slight, and one no improvement.

In about one-third of those showing striking improvement, antibiotics were given from one week to three months. In the remaining two-thirds, insufflation therapy was carried on from four and one-half to 10 months with the additional use, in some, of Gantrisin and aqueous antibiotics. All but five of the strikingly improved group of patients (61%) responded without further need of antibiotic therapy during the follow-up period of several months to more than one year. Priscoline and niacin were used throughout in combination except in one patient, where histamine was added.

In the significantly improved group, eight of the 10 patients were still under active treatment from two weeks to over one year. Priscoline and niacin were being given to some up to four months and others between four and nine

months. In a few patients, these drugs were taken continuously for about one and one-half years, the longest in one case being approximately two years, with the additional use of histamine most of the time.

The follow-up cultures taken in this later series of cases showed no growth of either Friedländer's bacillus or B. coli; however, H. staphylococcus aureus was predominant, except in two patients where B. proteus and odor persisted.

DISCUSSION.

Incidence: There is no specific information available on the morbidity of the disease throughout the world. Recent estimates obtained from various colleagues, listed in the table below,[41] as to the incidence in their clinic and private practice during the past five years offer reasonable evidence of a high prevalence throughout Europe, particularly in the southern and eastern parts as well as the Near East. Gusic has observed an even greater incidence in Yugoslavia since World War II as compared with the number of cases seen up to 1950. Several of our resident house officers from the Near and Middle East state that the disease is especially prevalent among their desert and agrarian populations. Ersner[36] reports a similar incidence. Wodak of Israel, however, writes that the number of ozena cases among the natives is extremely small by comparison with the European immigrants in the last few years. The impression throughout England, France and the Americas, is that it has shown a steady decrease in these countries during the past 25 years. In the absence of known causes, various explanations given are at best speculative. Pollock (1934) believed it was due to a restriction of immigration from Southern and Southeastern Europe; also earlier treatment plus prevention of chronic sinus infections as exciting factors. Quevedo of Guatemala asserts the cause to be late hereditary syphilis and consequently on the decrease. Gusic, however, observes that ozena is practically non-existent in the Dinar Highlands of Yugoslavia where lues is known to be endemic. In Cuba, Basterrechia believes the diminishing incidence is due to the yearly increase of diphtheria immuni-

zation in children. In Peru, Raffo attributes it to the improvement in hygienic and living conditions. The statistical estimates thus far received† and their geographic distribution are shown as follows:

ESTIMATES ON INCIDENCE FOR THE PAST 5 YEARS (1947 to 1953)

	Region	Hospital	Private Practice	Comments
United States				
Blaisdell, I. H.	N. Y.		12	
Daly, J. F.	N. Y.	15*	3**	* Bellevue Hospital ** 1953
Ruskin, S. L.	N. Y.		15–25	
Ersner, M. S.	Pa.	22*		* Temple Univ. and Jewish hospitals. Prevalent in Slovak group.
Henner, R.	Ill.		30–40	
Lederer, F. L.	Ill.		1* 5* 20*	* From associates. One per 500 Chinese recruits. (L)
Proetz, A. W.	Mo.		60	More prevalent in indigent foreign born population.
Brown, L. A.	Ga.		11	
Simonton, K. M.	Minn.	5		1949, Mayo Clinic
Thornell, W. C.	Ohio	10*	3	* 1946, Mayo Clinic
Lierle, D. M.	Iowa	35		University Hosp.
Brown, J. M.	Calif.		5	
Fitzhugh, W. M.*	Calif.			* Occurs in 5% of practice.
Morrison, L. F.	Calif.		42	
West Indies				
Gros, J. C.	Cuba, Havana	29* 14* 6***	2	* 1925, Univ. of Havana Hospital. ** 1945 *** 1952
MacCormick, C.	Puerto Rico, Santurce	10	5	
Font, J. H.			24	
Pico, J.			15	
C. and S. America				
Quevedo, J.	Guatemala	276* 213*	127* 115	* 1943-47 Assumed cause late hereditary syphilis.
Raffo, E.	Peru, Lima	15*	5	* Hospital Obrero. Decrease due to better living conditions.

† A more complete list will be reported later pending further replies from other countries.

ESTIMATES ON INCIDENCE FOR THE PAST 5 YEARS (1947 to 1953)
(Continued)

	Region	Hospital	Private Practice	Comments
C. and S. America (Cont.)				
Romero-Diaz, E.	Argentina, Cordoba	8–9		Hospital Cordoba.
Tato, J.	Buenos Aires	10		
Gutierrez, C.		7		
Jahn, F. C.	Venezuela* Caracas			* Exceedingly rare—due to antibiotics. Assumed etiology—B. pseudo-diphtheria.
Kos, J.	Brazil, Rio de Janeiro		60	Decrease noted.
Tello, A.	Chile, Santiago	130*		* Men, 28; women, 102.
Europe; Near East				
Negus, V. E.	England, London		10–15	
Ormerod, F. C.	London	107*	26**	* 1952, Inst. of Laryngol.-Otology. ** 1944-48
Portmann, G.	France, Bordeaux	60		* Clinique d'Oto-Rhino-Laryngologie.
Ruedi, L.	Switzerland, Zurich	187		Kantonsspital
Meurman, Y.	Finland, Helsinki	116		OPD not included.
Zollner, F.	Germany, Frieburg	82		Universitats-Klinik
Demetriades, Th.	Greece, Athens	451*		* Clinic cases, 1943-53
Gusic, B.	Yugoslavia, Zagreb	134*		* 1950-53
Wodak, E.	Israel, Tel-Aviv			Extremely rare among natives as compared with European immigrants since 1939
Australia				
Pettinger, D.	Sydney, Camperdown	6*		* Royal Alexandra Hosp. OPD.
Africa	Johannesburg	60–100		Non-European Hosp. OPD.

Although these figures represent random samples, they are, nevertheless, of sufficient significance to indicate the scope and challenge of the problem.

Odor: Despite various hygienic precautions of snuffing salt water, daily nasal douches, and oily sprays previously taken for many years, all patients were acutely apprehensive of their odor, whether actually present or not. The fear of being rejected as social outcasts was sufficiently provocative to channelize many deep-seated anxieties into a variety of psychosomatic symptoms. Recognition of their emotional problems and constant reassurance were important considerations throughout the entire course of treatment.

Hygienic Management: Allergic-like reactions of nasal obstruction, sneezing, ear blockage and asthmatic chest symptoms were occasionally encountered with the use of powdered or liquid Caroid (6%). Complete intolerance to the drug was present in 4 per cent of the cases.

Local Antibiotic Therapy: In comparison with aqueous therapy, antibiotic insufflation resulted in longer follow-up periods without regression after their discontinuance. Allergic skin or mucous membrane reactions were less encountered than with the aqueous antibiotics. Streptomycin in high dosage (200 mgs. once daily) appeared to promote in some a vascular hyperemia and slight bleeding of the nasal mucosa which was readily controlled with smaller or divided doses, *i.e.*, 50 to 100 mgs. twice daily.

The prolonged use of Gantrisin in both local and oral form was considered in general, effective against the resistant B. proteus organisms. Bacitracin proved efficient in its action upon gram positive flora without side effects wherever aqueous therapy seemed indicated. In the earlier group of cases, penicillin sensitivity of the nasal and oral mucous membrane was observed in over one-third (38%) of the cases from its aqueous use within a few weeks of local treatment. Only two (15%) showed allergic sensitivity to aqueous streptomycin, one of these after the fifth month in combination with penicillin.

Vasodilator Therapy: The immediate vasomotor response varied in its duration from several minutes in some to one hour or longer in others. It consisted of either a hot flush or

a tingling sensation of the scalp. Most patients noted a gradual increase in their body warmth, particularly in the hands and feet. In one case not included in this series, an irregularity of the menses was observed with Priscoline, returning to normal after the dosage was reduced. The use of the vasodilator drugs proved more effective in combination than singly for promoting the circulatory response and secretory function of the nasal mucosa. An indication of tissue permeability was evident from the resulting local manifestations such as, an increase in the erectile-tissue filling of the inferior turbinates, greater moisture content of the nose, the partial return of the sense of smell, and the local effectiveness of antibiotic therapy. It was not anticipated that turbinal recovery would take place where an atrophy of many years' duration had finally resulted in marked skeletonization. Nevertheless, even in this advanced stage, atrophic cellular changes were not entirely irreversible, since their histological improvement was observed after prolonged therapy.

Surgical Measures: In two cases with marked deviations of the septum, replacement surgery was additionally performed with submucous cartilage implants filling in the concavity defect to increase resistance to nasal airflow and, thereby, condensation within the nasopharynx. Resorption of the implant resected from the upper part of the patient's septum was observed in one case.

Results: The highest percentage of patients with striking improvement occurred in approximately 75 per cent to 60 per cent between the second and fourth decades. The percentage dropped from 50 per cent to 20 per cent between the fifth and seventh decades. The figures are illustrated as follows:

Years	Total Number Patients	RESULTS			No
		Striking	Significant	Slight	
0–10	1	0	1	0	0
10–20	4	3	0	0	1
20–30	5	3	2	0	0
30–40	8	5	2	1	0
40–50	4	2	2	0	0
50–60	10	4	4	1	1
60–70	2	1	1	0	0
70–80	1	0	0	1	0

In the strikingly improved cases, the earliest elimination of odor, crusts and discharge became evident from three to eight weeks with antibiotic insufflation. A decrease in turbinal atrophy was usually apparent after the third month with vasodilator therapy. As might be expected, the response was much slower in patients with significant improvement and in those who had had previous sinus surgery.

Thus far, the evidence based on the results reported in this paper offers no particular etiology but rather suggests that ozena may be a target disease which viewed in terms of successful therapy postulates a sustained overall approach from various directions both local and systemic. Most of the cases indicated a combination of symptoms pointing to metabolic, nutritional, and hematological deficiencies.

SUMMARY.

A new method for the treatment of ozena is presented by combining the local insufflation of various micronized antibiotics, such as Streptomycin, Terramycin, or Chloramphenicol (Chloromycetin) with the multiple use of certain oral vasodilators, Priscoline and niacin. Dosage and administration are described, including the additional use of aqueous bacitracin, Streptomycin, or Gantrisin. Sodium lauryl sulfate, a surfactant, and beta lactose, an acid vehicle, are incorporated with the micronized antibiotics for their greater effectiveness.

The management of nasal hygiene with the combined use of lamb's wool plugs and liquid or powdered Caroid 5 to 6 per cent (papaine) is carried out as a supportive measure to increase nasal moisture and dissolve encrusted secretions, thus eliminating the need for nasal irrigations.

A total series of 35 selected cases observed over a period of three years are reported. The results show 17 (48.6%) with striking improvement, 13 (37.1%) significant, three (8.6%) slight, and two (5.7%) with no improvement.

Three case reports with X-ray and microscopic evidence of turbinal growth and regeneration of respiratory epithelium are presented. The literature is reviewed.

CONCLUSIONS.

The prolonged use of micronized antibiotics for insufflation therapy in ozena causes no deleterious effects and constitutes a safe procedure.

Vasodilator drugs prove more effective in combination than singly for promoting the circulatory response and secretory function of the nasal mucosa. Turbinal recovery precedes demonstrable X-ray changes.

The combined use of properly selected antibiotics and vasodilators offers a promising method of treatment for the effective elimination of odor and crusts and a reversal of the regressive changes in ozena.

ILLUSTRATIVE CASE REPORTS.

Case 2, Later Series: B. N. A 26-year-old male, was first seen in the E.N.T. Clinic (1945) with symptoms of "sinus trouble" since boyhood.

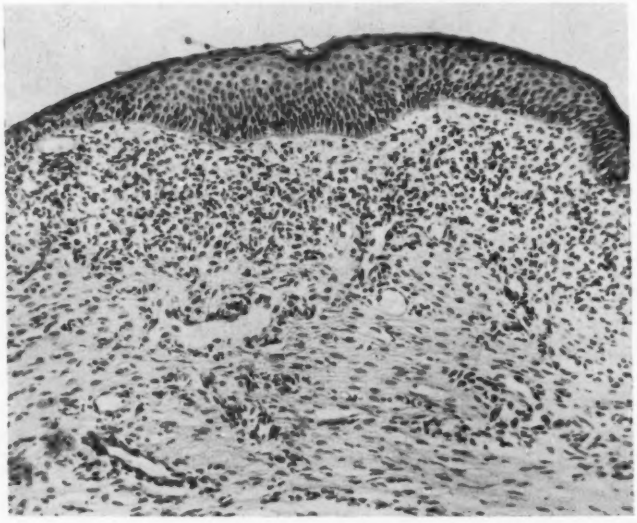

Fig. 2. B. N. Male. Age 26. Before treatment. Inferior turbinate (mid-third section) showing stratified squamous epithelium. Basement membrane and tunica propria appear within normal limits. Ill-defined subepithelial vascular spaces with no fibrosis. Infiltration moderate. High power. (130 X). Case 2. (Later series).

The nasal findings were recorded as odor, discharge and turbinal atrophy. There was a previous history of a submucous resection in 1938. X-ray of the sinuses showed pus in the right antrum and chronic thickened membrane in both ethmoids and antra. A right antrum wash disclosed a slight amount of pus. Nasal irrigations and an oily spray (iodine, menthol, camphor) were prescribed.

He returned for treatment six years later in April, 1951, and was referred to the atrophic rhinitis clinic for study. Examination revealed marked blockage, odor, green crusts, discharge, an anterior septal perforation (post-operative), moderate turbinal atrophy and anosmia. The culture reported was B. proteus. X-ray of the sinuses now showed no disease except for intranasal atrophy. A biopsy (see Fig. 2) revealed stratified squamous epithelium with a normal basement membrane. The lymphocytes and plasmocytes appeared within moderate limits and fibrosis of the tunica was not unduly dense. No glands were evident. The vascular spaces subepithelially appeared ill-defined showing neither fibrosis nor infiltration except for deeper thin-walled vessels.

Insufflation Therapy: Powdered Caroid (6%) and lamb's wool were prescribed for one month without appreciable effect. Insufflation with Streptomycin and later Chloromycetin, alternating with Gantrisin (oral and local), was carried on for over seven months.

Result: Striking clinical and histological improvement were noted with an increasing return of the sense of smell. A

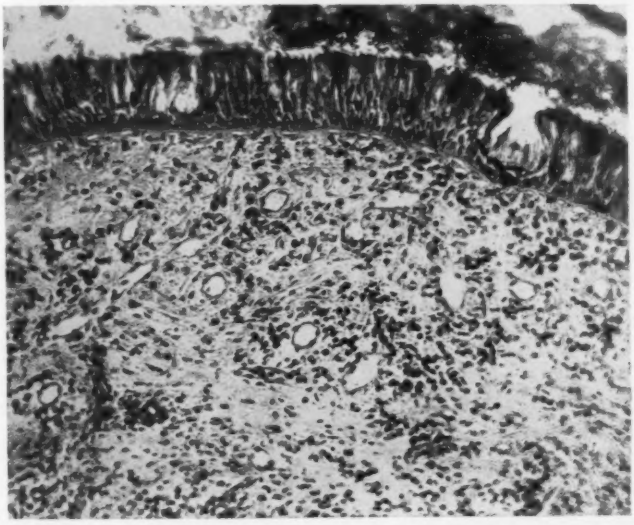

Fig. 2A. After 15 months therapy. Regeneration of respiratory epithelium with cilia and intraepithelial glands. Increased vascularity with many well defined capillaries superficially and larger vessels of normal appearance. High power, (130 X).

follow-up period for 10 months without antibiotics showed no regression. Priscoline and niacin, with the less frequent use of Caroid and lamb's wool was continued for a total of 15 months. Pneumococcus and H. staphylococcus aureus were reported in the last culture. X-rays taken after 14 months of treatment, June, 1952, revealed no demonstrable change in the atrophy. A biopsy (see Fig. 2A) after 15 months of therapy, July, 1952, showed regeneration of respiratory epithelium (Pseudo-stratified columnar ciliated epithelium) with intra-epithelial glands. The basement membrane and tunica propria appeared within normal limits as before except for a few newly observed glands. Many vascular spaces with well marked walls were seen as compared with their thin walled structure previously described. There were numerous capillaries and larger vessels of normal appearance.

Case 2, Earlier Series: P. C. A 53-year-old woman with a history of ozena dating back to childhood was first seen in the E.N.T. Clinic in 1937. Examination revealed odor, crusting, atrophy and anosmia. X-rays

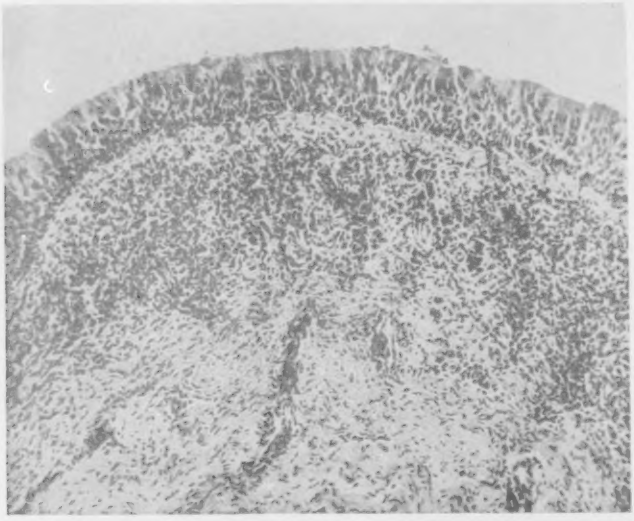

Fig. 3. P. C. Female. Age 53. After 12 months therapy. Inferior turbinate (mid-third section) showing respiratory epithelium, marked infiltration and fibrosis No goblet cells present. A few capillaries are evident but not well marked. Small blood vessels appear constricted by fibrous tissue. High power. (130 X). Case 2 (Earlier series).

of sinuses were negative. In 1938, a bilaterial Caldwell-Luc was performed for a bilateral chronic suppurative pansinusitis followed by estrogenic nasal sprays until 1946 with no improvement. X-rays still showed a bilateral chronic suppurative pansinusitis with marked intranasal atrophy. A revision bilateral Caldwell-Luc and intransal ethmoidectomy was again followed by a persistence of symptoms.

Combined treatment with aqueous streptomycin, histamine and niacin was started in February, 1950. Bacitracin was employed later due to penicillin sensitivity. Results after eight and one-half months of continuous therapy showed striking clinical improvement with complete inhibition of Friedländer's bacillus. Antibiotics were omitted for three months and vasodilators, lamb's wool and powdered Caroid (6%) continued. Tenacious secretions soon recurred in the nasopharynx, however, with no odor or crusting. H. staphylococcus aureus was reported in the culture. A biopsy (see Fig. 3) taken 12 months after therapy, showed respiratory epithelium. No goblet cells were present. The basement membrane appeared slightly thickened, and the tunica revealed marked evidence of chronic inflammation with lymphocytic and plasmocytic infiltration and fibrosis. Only one gland tubule was seen. There were many vascular spaces deep in the tunica propria with a normal number of small blood vessels constricted by fibrous tissues and a few capillaries not well marked.

Comment: Unfortunately a "before" biopsy was not obtained. (This also applies to the following case report, Case

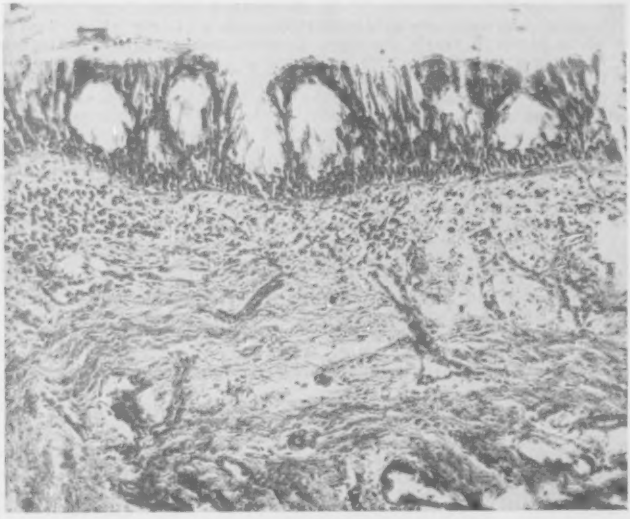

Fig. 3A. After 30 months' therapy. Definite regeneration of typical respiratory epithelium with goblet cells. Marked reduction of previous infiltration with increased vascularity of vessels close to the surface. Large spaces in epithelium are probably intraepithelial glands. Note well defined cilia. High power. (130 X).

13). The second biopsy, however, taken considerably later, showed further evidence of regenerative change.

Insufflation Therapy: Terramycin was prescribed for insufflation during the next three months.

Results: Striking clinical and histological improvement was observed with a return of the sense of smell. A follow-up period for 18 months without antibiotics showed no regression. Niacin, lamb's wool, and the occasional use of powdered Caroid were continued. H. staphylococcus aureus was last reported. X-rays taken in July, 1952, after two and one-half years of treatment revealed turbinal enlargement with a general reduction in atrophy. A second biopsy (see Fig. 3A) now showed definite respiratory epithelium with goblet cells and relatively little evidence of active infection in the tunica propria. One gland tubule was seen, also several new capillaries. The larger blood vessels appeared normal.

Case 13, Earlier Series: A. N. A 31-year-old woman was referred to the atrophic rhinitis clinic in April, 1950, with a history of ozena symptoms of 10 or more years' duration. Examination revealed considerable odor, crusting and anosmia with moderate atrophy. X-ray of the sinuses (see Fig. 1) showed marked atrophy of the turbinates but otherwise were negative. A culture was reported beta H. streptococcus, H. staphylococcus aureus and B. proteus.

Combined treatment was started with aqueous Streptomycin, Penicillin, Priscoline and niacin. Penicillin was omitted after one month because of sensitivity and aqueous Gantrisin 20 per cent was used more or less continuously for seven months without successful inhibition of B. proteus. After nine months therapy, there was significant improvement with clean nasal airways and normal appearing turbinates. An objectionable odor, however, was detectable due to persistent crusting in the nasopharynx. A biopsy at this time, showed both columnar epithelium and stratified squamous epithelium, corresponding to the lateral and medial surfaces respectively in the same specimen. The basement membrane in both appeared slightly thickened. There was slight to moderate cellular infiltration and fibrosis. The glands were slightly atrophied, and many small vessels showed perivascular fibrosis. Capillaries were scanty.

Insufflation Therapy: Chloromycetin insufflation was employed intermittently for nine months during the following year.

Results: Striking clinical and histological progress with a complete return of the sense of smell was observed over a follow-up period for eight months without antibiotics. The culture reported was a resistant H. stapyhlococcus aureus and B. proteus. Treatment was maintained with niacin, lamb's

wool and only the occasional use of powdered Caroid due to the blockage reaction produced. Priscoline was employed intermittently because of side reactions. X-rays (see Fig. 1A) taken in July, 1952, following 27 months of treatment showed turbinates of normal appearance. A second biopsy revealed a regeneration of respiratory epithelium. Goblet cells were not clearly identified. The basement membrane and tunica propria appeared normal. There were many small capillaries and larger vessels of normal appearance.

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MEETING OF THE GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Georgia Society of Ophthalmology and Otolaryngology will meet March 11 - 12, 1955, at the General Oglethorpe Hotel, Savannah, Georgia.

The speakers will be: Dr. Francis H. Adler, Philadelphia; J. W. McCall, Cleveland; Dr. J. A. Hilger, St. Paul; Dr. Walter H. Fink, Minneapolis; Dr. James H. Allen, New Orleans; and Dr. P. E. Ireland, Toronto, Canada. For further details, address ALTON V. HALLUM, M.D., Secretary, 245 Doctors Bldg., Atlanta 3, Ga.

HYDROCORTISONE ACETATE AND NEOMYCIN IN OTIC INFECTIONS.*†

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I. Introduction.—

In this era of new and remarkable chemotherapeutic agents and antibiotics, it is not unusual to find reports of excellent results in the treatment of recalcitrant otic infections; however, there have been few topical medications introduced in recent years which have proved of lasting value in the treatment of chronic and recurrent irritations in and around the external ear. Neomycin and hydrocortisone acetate (compound F) in proper vehicles seem to offer a solution to some of these difficult otologic problems.

Some of the most resistant infections and dermatoses found in the region of the external ear include simple pruritus or pruritus auris, localized neurodermatitis, postoperative infections of the skin lining the mastoid and fenestration cavities, and otitis media. These cases offer many problems as the physician attempts to control the primary infection as well as the secondary diffuse external otitis or infectious eczematoid dermatitis produced by the persistent drainage.

These dermatoses have been treated with some success with a variety of topical antibiotics and chemotherapeutic agents; however, these lesions are very prone to develop chemical or allergic reactions. In the mastoid and fenestration cavities, in particular, there has been a high incidence of fungus disease superimposed upon the original lesion as a result of the

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insufflation of various antibiotics; thus, some preparations have been helpful for a short period, but very few have demonstrated consistent success.

Radiation therapy is one of the techniques often used to control neurogenic lesions in and around the ears. This has offered excellent temporary improvement, but its frequent use is fraught with danger and, therefore, is used reluctantly by many otologists and dermatologists.

II. Literature Review.—

There have been many reports describing excellent results from the use of hydrocortisone acetate for various dermatoses.[1-5] In histopathologic studies of sundry skin inflammations it was demonstrated that compound F inhibited the inflammation of eczematous, toxic, and some miscellaneous skin disorders.[6]

Neomycin has been shown to have a marked bacteriostatic and bactericidal effect upon a wide range of bacteria,[7] including a variety of Gram-positive and Gram-negative organisms and Actinomycetes. It appears to be active against certain strains of E. coli[8] and Proteus[9,10] and is effective against some streptomycin-resistant strains of bacteria. Bacterial resistance develops more slowly than to other antibiotics, and no absolute resistance was demonstrated. On local application there is limited toxicity, but when given systemically it has been shown to be nephrotoxic. Neomycin is not effective against fungi or viruses.

Because of its broad coverage, neomycin in various water miscible and greasy bases has been used widely over the past few years for the topical treatment of a variety of dermatitis and dermatoses. It has been shown in comprehensive studies[11,12] that the therapeutic results with neomycin are superior to those with previous preparations. Patients with atopic dermatitis, stasis dermatitis, and seborrheic dermatitis were very tolerant to neomycin, but those with hemolytic streptococcus and pyocyaneus infections failed to respond satisfactorily.[11,13] In another study[12] a variety of conditions including impetigo, folliculitis, and impetiginized eczematous dermatitis were treated with impressive results.

Of particular importance is the low index of sensitivity described by various investigators. This preparation can be used in the highly sensitized areas without too much concern regarding secondary reactions even in those instances where reactions have occurred previously. One investigator reported localized cutaneous moniliasis following the application of neomycin;[13] another described a contact type sensitization apparently occurring five months or more after the beginning of treatment.[14]

Recently, Sulzberger and Rein[15] reported that hydrocortisone ointment topically applied to the ear was effective in relieving inflammation and itching in some cases of external otitis. Baer and Litt[16] suggested the use of a hydrocortisone-neomycin suspension and described the results in ten cases of external otitis of various and diverse morphologic and etiologic types.

III. Methods and Procedures.—

An accurate differential diagnosis of the type of external otitis present is essential, since it will be shown that this combination of drugs is very effective for certain types of inflammations of the external ear, while of questionable value in the treatment of other forms. Diseases of the external ear have been classified on an etiologic basis,[17] and cases are so divided in this study. Following are brief definitions of the various categories of diseases treated. Some overlap of categories is always present and will be commented upon later.

1. *Neurogenic External Otitis.* The largest number of cases in this category are classified as simple pruritus. This is predominantly an affliction of middle-aged women. Such persons are tense, hyperactive, and are very voluble about their sufferings. There is usually intense itching without visible lesions. They tug and scratch their ears and use any available instrument to relieve the persistent itching sensation. Cultures show a predominance of Gram-positive organisms although Gram-negative bacilli are seen occasionally.

   A small number of cases of localized neurodermatitis are included. These are characterized by various sized circumscribed, elevated, excoriated, scaly patches in

which there is thickening and an increase of skin markings. Other isolated plaques may be seen occasionally on the sides and back of the neck, eyelids, and anticubital areas.

2. *Diffuse External Otitis.* This is an acute or chronic symptom complex present mainly during hot, humid weather. The mild acute case has slight pain on manipulation of the auricle. The skin of the ear canal reveals some edema, slight redness and a coating of odorless, adherent secretion or exfoliated debris. In the more severe acute cases, patients complain of intense pain on mastication and on manipulation of the external ear. There is marked periauricular edema and partial or complete obliteration of the canal lumen by the edematous walls. Gray or green sero-purulent secretions and sheets of exfoliated debris are seen in the remaining lumen. The skin of the canal is thickened, purplish red in color and may have a papular appearance resembling gooseflesh, particularly on the superior and inferior walls.

   In the chronic case a diffuse thickening of the skin of the entire canal is found. There may be a fetid gray-brown or greenish secretion coating the skin and filling the tympanic recess. Papules and vesicles are sometimes seen, and the drum loses its luster, becomes thickened and shows some loss of detail.

   Cultures of the acute and chronic cases almost invariably show an abundant growth of Gram-negative bacilli although occasionally fungi are found. Stained smears reveal myriads of bacilli and epithelial cells.

3. *Postoperative Dermatitis of Mastoid and Fenestration Cavities.* In a small number of cases drainage persists from the postoperative mastoid or fenestration cavities despite active therapeutic measures. Cultures usually show a predominance of Gram-positive organisms.

4. *Otitis Media.* In this group are included subacute and chronic infections of the middle ear and possibly the mastoid antrum from which a mixed bacterial flora of Gram-positive and Gram-negative organisms is usually obtained.

5. *Otomycosis.* Diseases falling under this heading are chronic or recurring. The patient complains of intense itching, a feeling of fullness in the ear, difficulty in hearing and later, severe pain. Examination may reveal moist cerumen intermixed with exfoliated scales and sheets of epithelium, or the depths of the canal may be filled with wet grayish "blotting paper" debris. In uncomplicated cases the organisms may be seen as green, black, or white filamentous structures. Direct wet unstained smears reveal the presence of mycelia, spores, neutrophiles, and myriads of epithelial cells.

Hydrocortisone acetate ointment (1.5%)* was applied to all neurogenic lesions present in the concha, at the meatus or in and around the auricle. The patient was instructed to remove the adherent crusts or scales with mineral oil or vaseline and then to apply the ointment three times a day. If weeping and vesiculation were present, mild aluminum acetate packs were used as a cleansing astringent prior to the application of the ointment.

Neomycin-hydrocortisone acetate drops (1.5%) in an aqueous vehicle* were used in all post-fenestration and post-mastoid cavities and in patients with otitis media. Prior to the institution of therapy, the cavity was cleansed with hypertonic saline irrigations, and all exfoliated debris was removed by forceps or suction. Granulations were curetted away, and any evidence of fungi was treated with proper fungicidal agents. The patient was instructed to lie on the unaffected side, instill five drops of the suspension into the external ear for five minutes, three times a day. In occasional cases the ointment was applied to a wad of cotton which was then packed in the mastoid cavity and allowed to remain for 24 to 48 hours.

Irrigations were repeated on follow-up visits as often as necessary in order to keep the cavity clean and permit the therapeutic agent to come in close contact with the irritated

* Neocortef ointment containing 15 mg. of hydrocortisone acetate compound F and 5 mg. of neomycin sulfate per gram, and Suspension of Neocortef acetate (1.5%) containing 15 mg. of hydrocortisone acetate compound F and 5 mg. of neomycin sulfate per cc. were generously supplied by the Upjohn Co., Kalamazoo, Mich.

or infected skin. Cultures and smears were obtained both pre- and post-treatment as often as possible, and the bacteriologic changes recorded.

IV. Results and Discussion.—

The pertinent data and findings of the 90 treated patients (114 ears) are summarized in Tables 1 and 2. It is noteworthy that the average age of all groups is older than one would anticipate. There seems to be a predominance of females in the neurogenic and acute diffuse groups, but no predilection for one or the other ear.

The most significant clinical observation is the consistent and excellent response obtained in that group of indolent and recalcitrant ears placed in the neurogenic category. Only in occasional cases were indifferent results obtained.

The response of the patients was enthusiastic in that there was prompt control of the itching, vesiculation and weeping. Mild exacerbations were treated easily by the patient; thus the necessity for radiotherapy was eliminated in every case in this series. Needless to say, exacerbations occurred in those cases where the psychosomatic factors contributed to the production and persistence of the lesion. In these cases efforts were made to acquaint the patient with the underlying psychogenic basis for the dermatosis.

Of special importance to the otologist is the remarkable response obtained in the treatment of the persistently draining post-operative mastoid and fenestration cavities. Cavities which had been draining for years and showed ulceration and granulation suddenly responded to the application of this form of therapy. The skin lining which had appeared reddened, thickened and unhealthy, rapidly assumed a healthy color. Cavities which had shown granular, weeping islands, particularly on the tegmen mastoidea, responded promptly to treatment.

A number of unexplained failures with this form of therapy occurred in this latter group until previously unidentified fungi were found on smear or culture. Under these circum-

TABLE 1.

SHOWING DATA PERTINENT TO THE TREATMENT OF VARIOUS OTIC DISEASES WITH PREPARATIONS OF NEOMYCIN AND HYDROCORTISONE ACETATE.

Category	Average Age	No. Pts.	No. Ears	Ear Involved		Sex		Form of Medication		
				Rt.	Left	F	M	O	D	O & D
N.E.O.	40 yr.	22	32	16	16	22	10	21	4	7
A.D.E.O.	35 yr.	22	28	15	13	17	11	7	10	11
C.D.E.O.	48 yr.	6	10	5	5	5	5	1	4	5
O.M.	40 yr.	16	18	11	7	8	10	0	17	1
P.O.M.	24 yr.	9	9	2	7	6	3	0	8	1
P.O.F.	44 yr.	4	4	2	2	3	1	0	3	1
Otomycosis	26 yr.	8	9	5	4	5	4	2	7	0
Miscellaneous	20 yr.	3	4	1	3	1	3	1	0	3
Totals	35 yr.	90	114	57	57	67	47	32	53	29

N.E.O. —Neurogenic External Otitis.
A.D.E.O. —Acute Diffuse External Otitis.
C.D.E.O. —Chronic Diffuse External Otitis.
O.M. —Otitis Media.
P.O.M. —Postoperative Infection of Mastoid Cavity.
P.O.F. —Postoperative Infection of Fenestration Cavity.
Misc. —Perichondritis, Infantile Dermatitis, etc.

O—Ointment of Neomycin-Hydrocortisone Acetate.
D—Drops of Suspension of Neomycin-Hydrocortisone Acetate.

TABLE 2.

SHOWING DATA PERTINENT TO THE TREATMENT OF VARIOUS OTIC DISEASES WITH PREPARATIONS OF NEOMYCIN AND HYDROCORTISONE ACETATE.

Category*	No. Pts.	No. Ears	Predominant Bacteria		No Growth	No Culture	Exc.	Response		
			Gram-Positive	Gram-Negative				Good	Fair	Poor
N.E.O.	22	32	10	9	3	10	14	16	1	1
A.D.E.O.	22	28	4	16	1	7	11	12	2	3
C.D.E.O.	6	10	1	9	0	0	3	5	0	2
O.M.	16	18	9	6	1	2	10	5	0	3
P.O.M.	9	9	6	1	2	0	3	4	1	1
P.O.F.	4	4	2	1	0	1	2	0	1	1
Otomycosis	8	9	2	2	1	4	0	3	1	5
Miscellaneous	3	4	3	1	0	0	0	3	0	1
Totals	90	114	37	45	8	24	43	48	6	17

* See Table 1 for key.

stances all medication was stopped, and fungicides were applied for several days. Neomycin and hydrocortisone drops alone or a combination of drops and ointment was then used with rapid improvement.

The response to treatment of chronic otitis media cases was gratifying. The granulations appeared to shrink, revealing the periphery of the perforation and a view of the middle ear. In some of the cases it was then possible to nibble away a good part of the granulations. This allowed further penetration of the drops and further drying and shrinking of the middle ear tissues. In several cases large masses of cholesteatomatous debris could be removed with a small otic suction leaving dry middle ears.* It is to be noted that three failures occurred, and completely dry ears were not obtained in five others.

It was not anticipated that neomycin and hydrocortisone therapy would be highly successful in diffuse external otitis; therefore, in the early part of the study this type of treatment was used only when there was a secondary neurogenic element superimposed on the diffuse external otitis. The response was sufficiently good so that it was used for uncomplicated diffuse external otitis with relatively good results.

It is suggested that this is the treatment of choice wherever a question of differential diagnosis arises or where there are overlapping types. It is our belief that most antibiotics are contraindicated in the presence of a weeping erythematous lesion at the meatus. Whether primary or secondary to otitis media or diffuse external otitis, there is a high incidence of chemical or allergic reactions to "otic preparations" in these cases. In such instances, the safer combination of neomycin and hydrocortisone preparation appears indicated.

There are still many forms of external otitis which do not fall easily into clear etiologic or morphologic categories. Such cases have been placed in a small miscellaneous group. One of these, a severe perichondritis with superimposed pyocyaneus infection, responded nicely to packing with the ointment preparation.

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* Such ears will be observed carefully for possible recurrence because of the probability of residual cholesteatoma.

It is worthy of note that Gram-negative organisms, primarily E. coli and Pseudomonas, were cultured in more than half of the neurogenic ears. No streptococci were present among the Gram-positive organisms. It was not unexpected that almost all the cultures from the diffuse external otitis group showed predominant growth of Gram-negative bacilli.

The findings in this study confirm previous reports that cultures from the persistently draining postoperative mastoid and fenestration cavities usually show Gram-positive organisms.[18] Fungi are cultured in a surprisingly high percentage of these cases;[19] in fact, most of the patients listed under otomycosis are postoperative cases which responded poorly until culture reports revealed the presence of fungi.

The presence of a high percentage of Gram-negative bacilli in the cultures obtained from cases of chronic otitis media and mastoiditis is consistent with previous observations.[19] The excellent response of some chronic ears from which Proteus was cultured was not entirely unexpected in view of the reported *in vitro* effectiveness of neomycin.

In general this study points up the increasing necessity for the otologist and dermatologist to note clearly the differential diagnosis of diseases of the external ear. Only by appreciating the morphologic differences and the bacterial flora usually present in these various categories of diseases of the external ear can prompt and proper therapy be applied.

V. Summary.—

1. Ninety patients (114 ears) with various otic infections were treated with an aqueous suspension and/or an ointment containing neomycin and hydrocortisone acetate (compound F).
2. Consistent and excellent clinical results were obtained in the treatment of the neurogenic type of external otitis.
3. Recalcitrant infections of post-fenestration and post-mastoidectomy cavities showed remarkable response to the aqueous form of therapy.
4. The response of a high percentage of patients with chronic otitis media, acute and chronic diffuse external otitis was gratifying.

5. Otomycotic lesions failed to improve until the fungi were identified and eradicated.

We gratefully acknowledge the referral of post fenestration patients by Dr. Theo. E. Walsh and the technical assistance of Nancy Hellman.

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THE EVALUATION OF HEARING IN PRE-SCHOOL CHILDREN, WHO LACK NORMAL SPEECH. A PRELIMINARY REPORT.

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The child who has not learned to speak by the age of three years is commonly presumed to be retarded, or hard of hearing. There are, however, four major groups of children who have, in common, lack of normal speech at that age, namely, (1) The Deaf or Hard of Hearing; (2) The Brain Injured, including those with Aphasia; (3) The Autistic, or those with a severe psychic disturbance, and (4) The Mentally Retarded. It is obvious that an early diagnosis is essential for the proper medical, psychological and educational rehabilitation of these exceptional children. A qualitative method of differentiating these children, based not only on their response to sound but also on a careful analysis of their total behavior pattern, has been employed in a group study of 60 children at the Children's Memorial Hospital. The lack of normal speech was found to be due to deafness or impaired hearing in 50 per cent, and to other disabilities in the remaining 50 per cent of these children. A preliminary report on this study is herewith presented.

Hearing and Speech

Although deafness may be suspected early in an infant's life it is usually not until the child has failed to learn speech by the age of two or two-and-a-half years that the parents seek medical advice. A child may have other obvious disabilities, but the lack of speech immediately raises the question of whether or not the hearing is normal.

Normal Speech

Assuming the presence of normal speech organs, the development of speech depends upon a normal peripheral auditory mechanism, with properly functioning connections, *1*—to the

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auditory centers in the cortex (see Fig. 1). *2*—to the four sensory and motor speech areas, namely the frontal, temporal, parietal and supplementary areas which are usually present only on the left side of the brain (see Fig. 2), and *3*—to

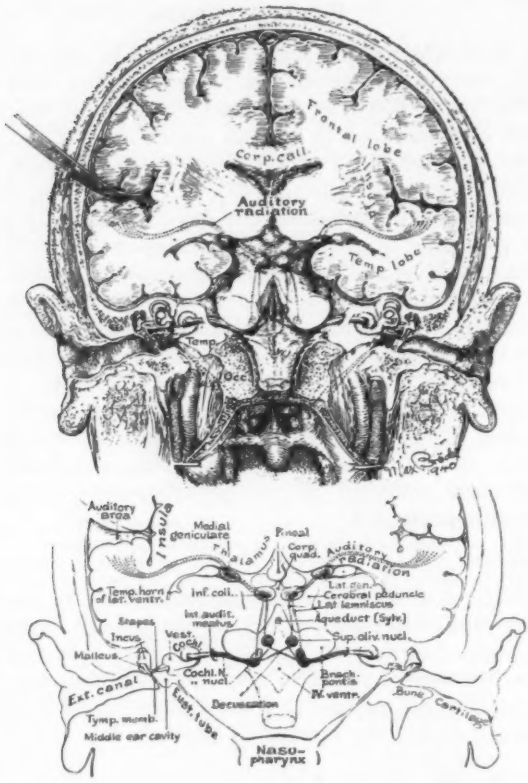

Fig. 1. From the Otological Research Laboratory and the Department of Art in Medicine, Johns Hopkins University, by the late Max Brödel (1940 Year Book of the Eye, Ear, Nose and Throat).

the centers for vocalization and articulation which are located on both sides of the brain (see Fig. 3). The connections between the auditory, speech, motor and other cortical areas

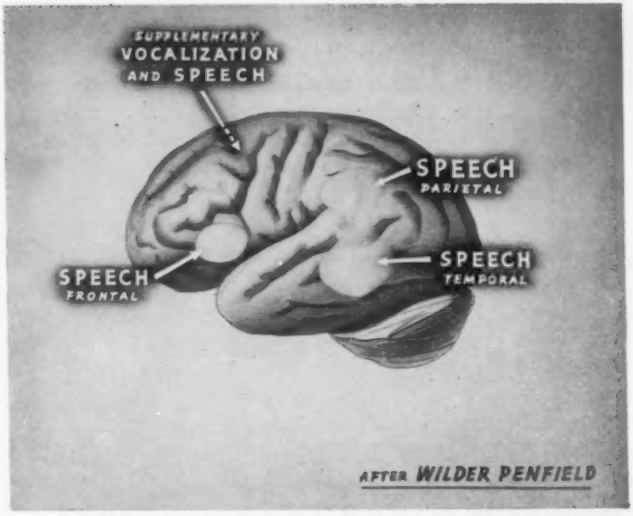

Fig. 2. After a drawing by L. Roberts (Localization of Speech in the Cerebral Cortex). 1951.

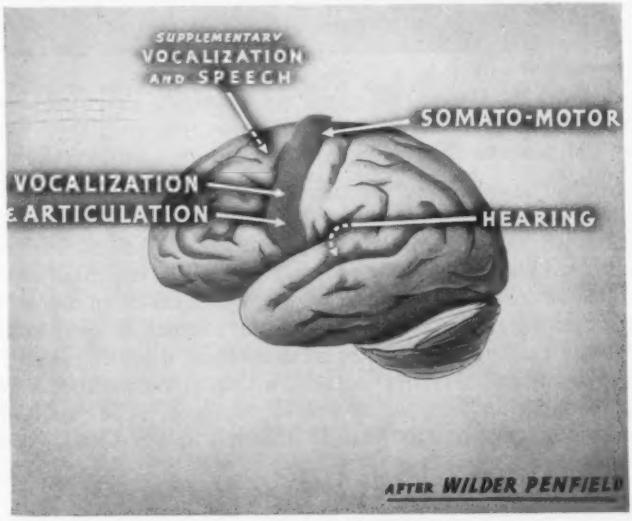

Fig. 3. Modified from a drawing in "A Consideration of the Neuro-physiological Mechanisms of Speech and Some Educational Consequences" by Wilder Penfield, 1953.

which are necessary for the reception, understanding and reproduction of the spoken word are all presumed to be subcortical (see Fig. 4).

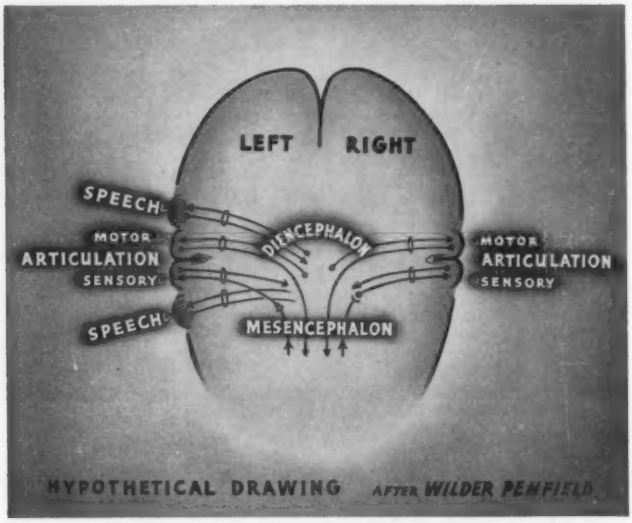

Fig. 4. Hypothetical diagram from "The Cerebral Cortex of Man," by Penfield and Rasmussen, 1950.

Lack of Normal Speech

Normal speech development, therefore, may be delayed or prevented by, *1*—a lesion in the peripheral auditory apparatus, such as congenital or acquired atresia of the external canal, with or without malformation of the ossicular chain, or disease in the middle ear, which would result in a conductive hearing loss; *2*—developmental anomalies or diseases affecting the organ of Corti in the cochlear duct, the ganglion cells, or the auditory nerve itself, which would produce a perceptive hearing loss, or complete deafness. The vestibular apparatus may, or may not, be involved in any of these conditions.

Failure of normal speech development may also be due to some central dysfunction, cortical or subcortical, with motor

or pyramidal tract involvement, as in cerebral palsy, or without motor involvement. In either case damage may involve one or more of the speech areas, resulting in motor or sensory aphasia. Minimal damage or dysfunction in the subcortical areas could also conceivably result in delayed or absent speech development.

Behavior of Handicapped Children

In considering children with speech disturbances it has been recommended by Myklebust that "rather than assess only the child's response to sound, the total behavioral pattern should be analyzed." He adds that "this approach has the advantage of recognizing that a certain child may not respond to sound for other reasons (than deafness), such as marked psychic disturbances, or he may respond inconsistently because of perceptual problems associated with brain injury." He emphasized that "the diagnostically differentiating characteristics should be those other than the ability or capacity to respond to sound." Hirsch stated that "the loss of hearing has ramifications that go beyond the mere loss of communicative skill to basic changes in the personality." The behavior of any child is the result of many factors, but the behavior pattern of the deaf child is basically the result of his disability, providing his disability is only the absence of hearing. Brain injured children show behavior disorders and perceptual problems peculiar to their disability, as described by Strauss and Lehtinen, while emotionally disturbed children may exhibit the peculiarities of infantile autism or schizophrenia described by Kanner. Many of the aforementioned children have been confused in the past with the mentally retarded. It would appear, therefore, that the behavior of these handicapped children is, at least in part, an expression of their disability or disabilities. There are cases that exhibit the features of two or more of these groups and these are the most difficult to differentiate at a time when a correct diagnosis is important for their rehabilitation.

Purpose of Study

The purpose of this study has been to apply the method established by Prof. H. R. Myklebust, and used in the Chil-

dren's Hearing and Aphasia Clinic at Northwestern University, to determine the major cause for the lack of normal speech in this group of 60 children.

Principles of Method

The basic principle of this method is to apply to the clinical practice of pediatric audiology that knowledge which is pertinent from the fields of psychology, psychiatry and special education. This method of investigation provides a realistic appraisal of the whole child. It recognizes the special problem of multiple handicaps, *i.e.*, impaired hearing and cerebral palsy, and it suggests that the child with more than one disability is best classified, for purposes of education and rehabilitation, on the basis of his major handicap.

Method Employed at Children's Memorial Hospital

The principles of this technique have been employed and expanded in this study over the past three years. The program at the Children's Memorial Hospital is primarily an inter-professional group study of each child. Information relative to every aspect of the child's problem is obtained from the detailed history, through ear, nose and throat examination, hearing tests, psychometric and speech evaluations. An electro-encephalogram has been obtained on each of these children, for evidence of focal or diffuse irregularities. When indicated, special blood studies, X-rays and consultations have been obtained with Psychiatry, Neurology, Ophthalmology and Pediatrics to make the investigation as complete as possible.

Steps in Differential Diagnosis

The first step in the differential diagnosis is a very complete history. This is obtained from the answers of the parents to a questionnaire similar to that prepared and used by Myklebust. This consists of a list of specific questions designed to obtain all possible information of etiological and diagnostic significance. This includes details of pre-natal, birth and post-natal conditions, history of all illnesses, operations and injuries, genetic development, emotional adjustment, auditory and language behavior. This information provides important differential clues as to whether the problem is organic or

functional and, if organic, whether it is peripheral or central. This data is obtained by the otologist before the child is examined or the hearing capacity tested, so that the hearing tests may be modified to conform with the type and age of the child under investigation. Valuable impressions may be gained if the child's behavior is observed during the history-taking period.

The second step is a thorough examination of the ears, nose and throat for evidence of disease or any condition which would affect the hearing, speech, or general health of the child. The integrity of the vestibular portion of the inner ear may be determined by caloric or rotation tests. This is important in all cases of suspected total deafness, in one or both ears.

In spite of the variety of methods used for testing the hearing of infants and pre-school children, accurate measurement of their hearing capacity still remains a difficult and challenging problem. Clinical experiences suggests, *1*—that the application of adult hearing test techniques to these young children is unsatisfactory, *2*—that pure tones may be too abstract and meaningless as auditory stimuli to brain injured children with perceptual disturbances, and *3*—that the child who fails to respond to sound immediately is not necessarily deaf.

The third and fourth steps in the differential diagnosis are combined. The hearing tests have been carried out in a sound-treated room with the child seated before a small table. While engaged in play with simple "educational" toys, many features of his behavior may be observed and noted; his general adaptability and his attitude toward strangers; his response to simple instructions by gesture or unexpectedly to verbal commands; his preference for the right or left hand; his ability to grasp and manipulate various objects, build simple structures, etc.; in brief, his general physicial and motor development, sensory and perceptual alertness, vocalizing ability, and emotional stability may be evaluated. The information obtained from the answers to the questionnaire may thus be correlated with the clinical study of the child during the hearing test period.

Behavior Characteristics

Inasmuch as the basis of this method is to assess, not only the child's response to sound but also his total behavior pattern, it is necessary at this point to outline briefly the characteristics of each group. It must be emphasized, however, that there are many variables. Some children do not fit exactly into a specific pattern, often because they have multiple handicaps; nevertheless, there is a pattern of general behavior which is recognizable for each of these groups: the deaf, the brain injured, the autistic, and the mentally retarded.

The Deaf Child

The behavior of a deaf child is different from that of a child with normal hearing. The loss of the primary warning sense, hearing, increases the dependence on the other special senses; therefore, the deaf child is especially alert to vibrations and visible movements in the environment. Unable to hear and speak, he communicates and makes his wants known by using gestures and pantomime, because he has good inner langauge and understanding. Failure to make himself understood may give rise to frustration tantrums. His voice tends to be flat and toneless. He does not improvise or play with sounds. His motor development is good, and he tends to be noisy and hyper-active. The deaf child is in general intelligent, friendly and cooperative at this age. He is generally consistent in his day to day behavior and not too difficult to manage in the home, providing the parents are intelligent, understanding and affectionate in the handling of his problem. If he is not totally deaf he will respond to sound, like a normal child, when the intensity reaches his threshold of hearing. This pattern of behavior is characteristic of the congenitally deaf child. It has been used in this study as a frame of reference for those who are presumed to be deaf.

The Brain Injured Child

The brain injured child is different in every respect. As a result of the brain damage caused by trauma, toxins or infection before, during or after birth, he may have disturbances of perception, concept formation, language and emotional con-

trol which are reflected in his behavior (Strauss and Lehtinen). These subtle expressions of brain injury may or may not be associated with the type of motor involvement seen in cerebral palsy. From our present point of view his basic disability is abnormal perception. He may misidentify visual, auditory, tactile and kinesthetic stimuli. Because all stimuli have equal power to attract him, he is distractible, hyperactive and inconsistent. Because the usual control, or inhibiting effect, exercised by the higher centers of the brain over the thalamus and hypothalamus is lacking, his emotional behavior is unstable and exaggerated. He is disinhibited, fearless, happy and affectionate. He is given to violent, uncontrollable outbursts of temper. He is destructive without anger. He is meticulous and pays unusual attention to minute details. He may be very friendly and intelligent, but he is inconsistent in his day to day behavior. He has a tendency to persist in repeating an activity once begun, long after it has ceased to have any meaning or purpose. This characteristic is called perseveration. He is difficult to understand and manage in the home, because he is so unpredictable and disorganized. Although he may be presented as a retarded child or behavior problem, his disturbed language development raises the question of whether or not his hearing is normal. His response to sound is inconsistent and at times remarkable. He may not respond to pure tones presented in the free field at intensities approaching the threshold of pain, and yet a moment later definitely respond to a whispered voice or the squeak of a toy rubber mouse, depending upon his attention at that moment.

The Autistic Child

The autistic child is very strange. He may be confused with the feeble-minded or, because of his muteness and inadequate responses to sound, with the deaf. The behavior characteristics of this severe form of psychic disturbance were first described by Kanner in 1943 and called infantile autism. This type of child is withdrawn, non-communicative, self-sufficient, happiest when left alone, meticulous in details, adverse to changes in routine, interested primarily in objects and not people, not even his own parents. He treats people

as objects, shows no affection, and will rarely look anyone in the face. These children are intelligent, but their "cognitive potentialities are masked by their basic disorder," which has been defined as "a disability to relate themselves in the ordinary way to people and situations, from the beginning of life." The child's behavior is "governed by an anxious obsessive desire for the maintenance of sameness that nobody but the child may disrupt" (Kanner). He is given to panic tantrums, followed by sudden calm. His face is placid, but occasionally he may be found smiling, humming or singing to himself. Hebephrenic and catatonic types of schizophrenic behavior may also be observed in the most seriously disturbed children. The hebephrenic type is characterized by curious mannerisms, gesticulations, tic-like movements, and unexpected spells of laughing and crying. Compulsive and repetitive actions are common. The catatonic type is more generally tense, rigid, and contrary. Awkward positions may be maintained for long periods. The autistic child pays no attention to his surroundings. He may not respond even to a painful stimulus. He may appear to be in a stupor. Here, again, it is the lack of speech, and absent or inadequate response to sound, which may lead to a suspicion of deafness. The child's characteristic pattern of behavior, however, provides a clue to the basic disability. An accurate assessment of the hearing capacity is most difficult because of the child's self-imposed isolation and lack of normal response to any stimulus. The pathology of this condition is unknown. Although some of these children may remain mute, the majority apparently have normal hearing.

The Mentally Retarded Child

The mentally retarded child performs at the level of his intellectual or mental maturation. He is retarded in all phases of his development, including language and perception. He is unable to take care of himself. His social, learning, and mental age do not relate favorably to his chronological age. The various types of idiots, morons, feeble-minded and so-called inadequate children can be differentiated by specific tests which are best applied by those who are qualified, by special training, to interpret the results in terms of educability of

the child in question. If there is a suspicion of a hearing impairment, the type of hearing test employed must be in keeping with the mental age of the patient under consideration.

Objective of Hearing Tests

The primary objective of these hearing tests has been to determine whether or not the child who lacks normal speech has adequate hearing to learn speech. For this purpose it is sufficient to determine that his unaided hearing is within the zero to 30 decibel zone for the speech frequencies.

Hearing Test Equipment

The hearing test equipment includes (1) noise makers, such as tin crickets, rubber mice, sleigh bells, rattles, whistles, etc., and (2) children's rhythm band percussion instruments, including a metal triangle, cymbal, "A" chime, tambourine, wooden tone blocks and castanets. The intensities (at one and three feet) and the frequency range of all these have been determined as accurately as possible at the National Research Council Laboratories, Ottawa. With an Octave Band Pass Filter these "calibrated" noise makers were found to have peak frequency ranges as follows: tambourine, 200 to 800 cycles per second when the drum part was tapped, and 3,200 to 6,400 when the instrument was shaken; wooden tone block, 800 to 1,600 cps.; large cricket, 1,600 to 2.400 cps.; small tin cricket, 3,200 to 6,400 cps.; sleigh bells, 2,400 to 9,600 cps., and a squeaking rubber mouse, 6,400 to 8,600 cps. The intensities range from 20 to 100 decibels at one foot. (3 Pure tones from an audiometer are presented through a loudspeaker situated three feet away from the child. (4) Recorded "familiar" sounds are also presented through the loudspeaker into the sound field. These include the sounds of a train whistle, vacuum cleaner, telephone, aeroplane, auto horn, cat, dog, cow, bird, bell, etc., and the voices of a woman, man and child. All of these may be presented at controlled intensities. (5) Simplified live voice tests may be used, when indicated, to determine a child's capacity to hear and understand speech. An approximate speech reception threshold may thus be obtained. (6) Recorded music has been employed in cases of suspected aphasia when the responses to the other tests

were inconsistent but suggested that the hearing was adequate to learn speech. In several such cases the child danced to the music when it was presented at an intensity level well within the limits of normal hearing. This response was repeatable and, as far as could be determined, not due to the feeling of vibrations.

The child's vocalizations provide valuable information. If an infant will imitate humming sounds, or repeat baa, baa, mom, mom, after the examiner, there is definite evidence that he heard these sounds. This vocalization test is very useful for the brain injured or mentally retarded child, to demonstrate that he responds at a conversational level.

Hearing Test Technique and Response

As mentioned before, these objective hearing tests have been carried out in a sound treated room, with the child seated at a small table. While an assistant keeps the child occupied in a play situation with toys, the examiner activates the calibrated noise makers at a distance of three feet behind the patient, beginning with those of the lowest intensity. If no response is obtained, the tests are repeated at a distance of one foot behind the patient, or at either ear. Although great care is taken to keep the noise makers out of sight during these tests, they may be brought into the periphery of the child's visual field deliberately to note his reaction. Deaf children are usually very alert to movements in their environment, whereas children with severe psychic disturbances may completely ignore the sound makers, even when the instruments are struck directly in front of them. Pure tones and recorded familiar sounds are then presented in the free field at increasing intensities. In our experience, children of the pre-school age group do not tolerate head phones very well. hence the almost exclusive use of the free, or sound, field method. The most commonly observed response to these auditory stimuli is cessation of activity, with head turning to identify the source of the sound. A startle response, reflex eye blinking, or eye turning may be observed when loud sounds are presented to young infants. It is noteworthy that once a response is obtained the child, even with normal hear-

ing, may not respond again until a different or stronger sound stimulus is presented.

It is very difficult to assess accurately the hearing of brain injured children with the type of motor involvement seen in cerebral palsy. They have to be "structured" in their hearing tests by eliminating all other stimuli and by having them visually identify and play with the sound makers before they are presented as auditory stimuli. In this way responses may be obtained in otherwise apparently deaf children.

Interpretation of Responses

The child's response to sound is interpreted in relation to the behavior pattern observed during the hearing tests. The history, physical examination and clinical analysis of the behavior pattern are thus correlated and used to validate the responses, or lack of responses, to the hearing tests. The technique of applying these hearing tests has been standardized, and an approximate audiogram has been obtained on all of the children in this study.

Classification of Hearing Test Results

Regardless of the child's major handicap, his hearing has been classified as follows:

1. *Adequate to learn speech,* when he responds to the auditory stimuli mentioned above, at intensities of 30 decibels or less, in the speech frequencies.

2. *Impaired hearing for speech development,* when he responds to stimuli at intensities of not less than 35 to 65 decibels. This degree of hearing loss is usually due to a conductive lesion, and often it can be raised to a useful level by amplification or treatment.

3. *Inadequate to learn normal speech,* when he fails to respond to stimuli of less than 70 decibels. This is most commonly due to an inner ear or perceptive lesion, with or without a conductive component.

4. *Deaf,* when he fails to respond to any of the sound stimuli.

This classification may then be used as a guide in planning the child's medical and educational rehabilitation program.

Correct Diagnosis

In evaluating a child suspected of having impaired hearing by this qualitative method, the diagnosis is based on the history, physical examination, hearing assessment and analysis of the behavior pattern. This method has proven to be practical in differentiating the deaf child from those children whose lack of speech is due to some form of central damage or dysfunction.

A correct diagnosis is obviously necessary for the proper education, support and guidance of the parents of these children. They need assistance and encouragement in the understanding and handling of their problem in the home.

The necessity for a correct diagnosis is emphasized in outlining a program of medical, psychological and specialized educational rehabilitation for each of these exceptional children, since each of the four groups mentioned requires a different type of rehabilitation program.

RESULTS.

The average age at which these children were first examined was 31 months. They were all suspected of having impaired hearing because of their speech deficiencies. It was found that 25, or 42 per cent of this group suffered multiple handicaps. Classified on the basis of their major handicap, for purposes of medical and educational rehabilitation, 30, or 50 per cent, had insufficient hearing to learn speech because of some peripheral auditory disturbance. Of these, 14 were deaf, 14 had a very severe hearing loss (of 80 decibels or more), and two had a severe hearing loss (of 60 to 80 decibels).

The remaining 50 per cent were considered to have adequate hearing to learn speech. Their lack of normal speech was apparently due to:

1. Brain injury in 29 per cent of the total 60 children. One-third of these children had cerebral palsy. The diagnosis of brain injury was confirmed in the majority of the remaining cases by an abnormal electroencephalogram.

2. Mental retardation in 13 per cent of the total. The degree of retardation was established by the psychometric evaluation.

3. Severe emotional disturbances in 8 per cent of the total e.g., autism and schizophrenia. This diagnosis was confirmed by the psychiatric consultant.

CONCLUSIONS.

1. The purpose of this study has been to evaluate the method of investigation recommended by Myklebust for children who are suspected of having impaired hearing because of their lack of normal speech.

2. This method has been employed and expanded at the Children's Memorial Hospital by organizing an inter-professional diagnostic group. This consists of representatives from otology, audiology, speech therapy, psychology, psychiatry, neurology, pediatrics, ophthalmology, electroencephalography, and social service. The specialized knowledge of these consultants has been applied to the final assessment of each of these handicapped children, and utilized in proposing a program of rehabilitation.

3. This qualitative method has proven to be practical in differentiating the deaf child from those children whose lack of speech is due to some form of central damage or dysfunction.

4. Although the elaborate plan of total investigation described in this paper was organized for a specific purpose, the principles of this method may be applied by the otologist as a valuable diagnostic guide in clinical practice. He may thus be assisted in planning the correct program of investigation or rehabilitation of these handicapped children.

ACKNOWLEDGEMENTS.

The authors gratefully acknowledge the assistance and cooperation received during this study from Dr. Arthur Cohen, Dr. H. Jasper, Dr. A. Hefferman, Mrs. A. Farkas, Mrs. H. Golick, Mrs. E. Duschenes, Dr. P. Robb and Dr. W. Laws.

The authors also thank Dr. T. D. Northwood, Division of Building Research, National Research Council, Ottawa, for his cooperation in calibrating the noise makers and percussion instruments.

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I CONGRESO EXTRAORDINARIO DE LA SOCIEDAD INTERNACIONAL DE AUDIOLOGIA.

Buenos Aires - Republica Argentina.

The First Extraordinary Congress of the International Society of Audiology, with headquarters in Paris, will be held in Buenos Aires, November 26 - 30, 1954.

This important scientific gathering held outside France for the first time, will be honored with the presence of prominent international scientists, specially invited, from the United States, France, Belgium, Switzerland, Holland, Britain, Germany, Brazil, Chile and Paraguay. It has also been supported by all the Universities of the country, of well known local and foreign centers of studies, as well as of delegates from several European and American countries.

For further information address Dr. Atilio Viale del Carril, or Dr. Renato Segre, general secretaries, Buenos Aires, Argentina.

LARYNGEAL COMPLICATIONS OF ENDOTRACHEAL ANESTHESIA.*

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It is generally recognized that endotracheal intubation has now assumed a permanent place in anesthesia. Because of the universal use of this method of administering anesthesia it is important to recognize the accompanying hazards and contra-indications to such a procedure. Such recognition will, by our awareness of them, eliminate many of the problems. This paper discusses the basic etiologic factors, diagnosis, and treatment of these complications.

The careful observer can elicit symptoms and see objective findings indicating laryngeal pathology in as high as 50 per cent of the cases in which endotracheal intubation was performed if he records all minutia; however, there is a relatively low incidence of persisting serious pathology requiring active treatment. It is important to remember that serious complications do occur and must be recognized early to prevent disability or even death. The specific problems with which we must concern ourselves range from submucosal hemorrhage, abrasions of mucous membranes, contact ulcers and granuloma, membrane formations, subglottic edema, lacerations, to perforations of the tracheo-bronchial tree or esophagus.

ETIOLOGY.

The etiologic factors may be divided into two parts: the first, the intrinsic factor, refers mainly to the normal anatomy of the hypopharynx, larynx, and trachea. The anatomy is such that we can anticipate the points of stress and trauma. The action of the larynx is to protect the tracheo-bronchial tree as well as to act as an organ of phonation, and as such, it has a powerful sphincteric reflex which is activated at the slight-

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est stimulus. This results in considerable trauma to the intrinsic larynx during severe cough and spasm. There is a constant motion of the arytenoids with respiration which results in continuous trauma to the cords, even under anesthesia, should a foreign body such as an endotracheal tube be in place. The vocal process of the arytenoid projects into the vocal cord and is covered only by a thin layer of perichondrium and mucosa. This makes it extremely responsive to trauma.[1] The pyriform sinuses with the cricopharyngeus muscle guiding the entrance to the cervical esophagus are easily accessible to perforation. The laryngeal ventricles are so placed that the endotracheal tube will enter into the ventricle rather than the trachea should the bevel be turned to the side or the cords be in spasm. The subglottic area with its loose areolar tissue quickly becomes edematous after trauma. All of these are normal anatomical facts.

We must also keep in mind the abnormalities of these structures due to super-imposed intrinsic factors. This includes post-irradiation edema and dryness of the mucous membrane. Also chronic infection of the mucosa results in a loss of ciliary activity, increased friability, and desquamation of the mucous membrane. All of these add up to an increased sensitivity to trauma.

When considering the extrinsic factors in the etiology of laryngeal complications, number one on the list must be the technique of the operator. Long ago, Dr. Chevalier Jackson described the proper technique for intubation of the larynx.[2] It has not changed over the years. In endoscopy there is no place for the heavy hand. It will be agreed, I am sure, that direct vision has an advantage over blind intubation in avoiding trauma. There must be a careful choice of instruments insofar as size and condition are concerned. This will be discussed more fully later. The proper plane of anesthesia must be obtained to avoid spasm and resistance to the passage of the tube. It is important to maintain this proper plane of anesthesia to avoid coughing, swallowing and other factors causing continued motion of the tube or of the larynx on the tube. Finally, it is important to avoid makeshift apparatus.

DIAGNOSIS.

The diagnosis of laryngeal complications is made on the evaluation of the patient's complaint plus direct examination and objective findings as shown by the laryngeal mirror or laryngoscope.

Laryngeal complications can be divided into two basic groups: there are those which occur immediately or within the first 48 hours. These include abrasions, submucosal hemorrhage, edema, laceration, and membrane formation; then, there are those which may develop from a few days to six months later, such as ulceration and granuloma formation. All of these patients will complain of a sense of discomfort in the throat which may become severe enough to be described as actual pain. Hoarseness is also present in nearly all cases.[3] These complaints, however, when associated with progressive stridor, dyspnea, restlessness, anxiety, tachycardia, sternal retraction, and finally anoxia and exhaustion, call attention immediately to an obstructive phenomenon in the upper respiratory tract. In nearly all cases this will be due to laryngeal edema or to the formation of a subglottic membrane.[4] While they occur most frequently in infants and children, they can also occur in adults. The presence of a subcutaneous emphysema is indicative of a perforation. Severe dysphagia may be associated with perforation but is also present following traumatic laryngoscopy. X-rays, of course, are important for those cases of suspected perforation. In the last analysis, the diagnosis of laryngeal pathology is confirmed by mirror laryngoscopy and/or direct laryngoscope. Treatment should be instituted immediately as indicated. Care must be taken during mirror laryngoscopy to avoid missing the subglottic stenosis or obstruction since the supraglottic structures may be normal in appearance. Negative mirror laryngoscopy, in the presence of positive signs and symptoms, indicates the need for direct laryngoscopy, tracheoscopy, or bronchoscopy.[5]

TREATMENT.

Treatment must be divided into two phases: the first is the prophylactic regime which is carried out both by the anesthesiologist and by the attending surgeon or physician. Pre-

operative elimination of chronic infection in the sinues, oral cavity and pharynx and tracheo-bronchial tree by chemotherapy is valuable. Again we must reiterate the need for gentle, atraumatic, carefully executed intubation with proper instruments. Not only is the proper size of the instrument important, but cleanliness is also a factor too often neglected. Introduction of a virulent organism into an abrased mucous membrane is not to the best interest of the patient. Endotracheal tubes must not be allowed to become roughened by careless handling. Stylets are dangerous, and care must be used to avoid trauma to the anterior wall of the trachea or pyriform sinus. The lubricating agent may be irritating and cause a tissue reaction. Care should be taken to place the cuff well below the larynx, keeping it small and the surface smooth. Any tube which gives an air tight fit in the trachea without inflating the cuff is one which is liable to cause a subsequent edema.

The use of endotracheal anesthesia in children should be a matter of consultation between the surgeon and the anesthesiologist. We know that these are the cases where it is difficult to predict the reaction of the larynx to intubation; therefore, it would seem logical to avoid endotracheal anesthesia in such patients except where there is a definite need for it. The anesthesiologist should not become so dependent upon the tube that he fails to develop his own full scope of anesthetic techniques and thus be forced to use the endotracheal technique where it is not absolutely necessary.

Inhalation therapy, humidity, systemic chemotherapy, vocal rest, and avoidance of irritants such as talking and smoking are helpful in preventing further reaction to a possible traumatized larynx in the postoperative care of the patient following endotracheal anesthesia.

Direct treatment of these complications refers in the main to the endoscopic removal of granuloma or of the fibrinous exudate accumulating in the subglottic space, as well as to tracheotomy. While we try to avoid the need for direct treatment in a way described previously, should mirror or direct

laryngoscopy show the formation of a granuloma or fibrinous exudate, direct intervention is indicated. The granuloma of the vocal cords usually occurs in the posterior third of the cords and is often bilateral. This will often recur following direct laryngoscopic removal, and it has been found that the smaller the stalk of the granuloma, the less chance of recurrence. An occasional granuloma may become pedunculated to the point where it will be extruded by a sudden cough. This does not happen too frequently and one should not rely upon this method of treatment. Direct laryngoscopy with removal of the excess tissue followed by a strict regime of vocal rest and elimination of irritants is the indicated treatment when the diagnosis of contact ulcer granuloma is made. Where there is no granuloma, the contact ulcer will usually heal on a regime of vocal rest and speech therapy.[6] Of course, severe edema with obstruction of the airway may result in the need for tracheotomy. Be sure that it is true edema and not a membrane which can be removed by direct laryngoscopy. Tracheotomy should be carried out early to avoid fatality.[7] There is no question that in this instance procrastination is dangerous. The safe rule to follow is that when one begins to debate the need for tracheotomy, then is the time to do it.

Finally, a word about division of responsibility in these problems. There must be a close coordination between the laryngologists and the anesthesiologists. Responsibility must be clearly defined. The person called for the emergency consultation must be equipped to carry through the immediate examination and treatment; that is, mirror laryngoscopy, direct laryngoscopy, intubation, and tracheotomy. Proper equipment must be available for use in such emergencies. There is a point in the examination of these patients, particularly in children with obstructive membrane formation or edema, where one must proceed with the removal of the membrane or insertion of an endotrachial tube or bronchoscope, or the resultant spasm and reduction in airway may result in asphyxia of the patient. It is dangerous to take a look at a larynx with a direct laryngoscope in patients with obstructive phenomenon without having available the endoscopic or tracheotomy equipment to re-establish the airway quickly.

CONCLUSION.

I have discussed the underlying factors in the etiology, diagnosis, and treatment of laryngeal complications of endotracheal anesthesia. We must make every effort to reduce them. Endotracheal anesthesia is here because there is a real need for it. We must progress in turn and establish the techniques which will reduce these complications to a negligible degree. This can be done by proper attention to the basic concepts of anatomy, physiology, and endoscopic technique.

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230 North Broad Street.

BRONCHOESOPHAGOLOGY COURSE.

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for November 8 - 20, 1954. Under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

COTTON GRANULOMA OF THE MASTOID.*

CASE REPORT.

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Baltimore, Md.

The following case presents a most interesting and unusual aspect of chronic middle ear disease.

HISTORY.

The patient, J. R. G., a 69-year-old white male, dependent of an officer, was seen by the Department of Otolaryngology at the School of Aviation Medicine in November, 1952, with the complaint of vertigo, tinnitus, headaches, cervical pain, and a discharging left ear. His difficulty began when he was six years old. Following an attack of measles the patient developed bilateral acute suppurative otitis media. He recovered from the measles, but the ears continued to discharge, and this condition progressed into chronic suppurative otitis media. The patient consulted an otologist who treated and eventually cleared up the discharge from the right ear; however, the left ear did not respond to treatment and continued to suppurate throughout his adult life. At the age of 37, the patient was advised to undergo a mastoidectomy as a possible means of stopping the discharge. He consented and a simple mastoidectomy was performed on the left ear at that time. Postoperatively, the patient's condition improved to the extent that the amount of discharge diminished but did not cease. Five years after the operation the patient experienced severe attacks of vertigo followed by nausea. He sought the advice of a gastroenterologist who, after an examination, suggested that he consult an otologist concerning his ear. The otologist instructed the patient to keep the left ear clean and dry. Since that time the patient has meticulously cleaned his left ear with a dry cotton swab every day. In 1949, the patient was hospitalized because of a genito-urinary complaint and, while in the hospital, had an otolaryngologic consultation because of his chronically draining left ear. Examination at that time disclosed a large polypoid mass in the left middle ear. This was removed by means of a snare through the external canal. Microscopic studies of this tissue revealed it to be a polyp with excessive fibrosis. The patient was somewhat improved after this procedure, except for the persistent discharge and occasional attacks of tinnitus and vertigo. For the past six months he has been suffering from generalized headaches and cervical pain, with spasm and twitching of the left side of the face. His hearing has been impaired in both ears since youth, and he has worn a bone conduction type hearing aid on the right ear for 35 years.

Past History:

The patient had a tonsillectomy and an adenoidectomy when a child. A submucous resection of the nasal septum was performed when he was 32 years old. He has no known allergies or sinusitis.

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* From the Department of Otolaryngology, USAF School of Aviation Medicine and Department of Radiology, Randolph Air Force Base Hospital, Randolph Field, Texas.

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EXAMINATION.

Examination of the right ear revealed the external canal to be normal. The tympanic membrane was scarred, retracted and distorted. There appeared to be a small perforation in the anterior, inferior quadrant, in which a slight amount of mucoid discharge was present. Upon examination of the left ear a well healed postauricular scar and a slight pit-like depression in the left mastoid process was noted. The external

Fig. 1. Lateral view of the left mastoid. A large and fairly distinct area of erosion is evident adjacent and posterior to the canal.

canal was normal. The tympanic membrane was absent. There was a large pulpy mass that filled the middle ear cavity and extended to the level of the tympanic ring. Purulent material was observed around this tumor-like mass. A biopsy of the mass was taken. The nose, throat and nasopharynx were essentially negative. Bone conduction was good in the right ear and fair in the left. Air conduction was poor in both ears. Roentgenograms of the temporal bones (see Fig. 1) revealed the

presence of a few air cells in the right mastoid amid considerable sclerosis. The few cells present were fairly distinct, and there was no suggestion of active inflammation. On the left there was a large punched-out defect posterior to the auditory meatus which had sharp, well-defined edges. No definite air cells could be demonstrated. The mastoid process was found to be grossly sclerotic and in addition showed evidence of an old mastoidectomy. The roentgenographic evidence of erosion suggested the presence of a neoplasm. possibly arising in an area of squamous metaplasia. Cholesteatoma was also considered, but the edges of the eroded area were not as ragged and indistinct as they might be if the bone were being destroyed by a malignant neoplasm or destructive mastoiditis. Abscess formation is less common than cholesteatoma in the sclerotic mastoid.

The pathologic report of the biopsy of the mass in the left ear revealed it to be a granuloma resulting from chronic inflammation. There was no evidence of malignancy present. The impression was: *1.* Pyogenic granuloma in the left middle ear and attic which produced erosion in the attic, secondary to chronic suppurative otitis media; and *2.* Chronic suppurative otitis media, right, quiescent. Because of the evidence of erosion in the left attic the patient was advised to undergo a radical mastoidectomy. This was performed on February 3, 1953.

Fig. 2. Cotton foreign body removed from the mastoid antrum.

OPERATION.

Under general anesthesia the left auricular area was prepared in the routine manner. A postauricular approach was used. The skin and subcutaneous tissue were incised and retracted, exposing the mastoid process of the temporal bone. Upon examination a large eroded area of bone approximately 1 cm. in diameter was found in the region of the antrum. This eroded area was covered only by periosteum. The periosteum was incised and retracted, and a large amount of granulation tissue and debris was noted in the antrum. This was removed by curettage and inspection revealed a hard, fibrous-looking mass that appeared to be firmly imbedded in the antrum. After careful dissection this mass was removed in its entirety and found to be an old piece of cotton (see Fig. 2) that persumably had been left in the antrum at the time of the

previous surgery over 30 years ago. This cotton measured 9 x 12 mm. Exploration of the attic disclosed erosion through the tegmen, so that a small area of the dura was visible. The erosion also extended through the facial canal and exposed the VIIth Nerve. Granulation tissue and necrotic material were curetted from the middle ear and the attic. Rough and overlying bone was smoothed and removed with a gouge and curette; the tympanum, malleus and incus were not found. The mastoid cavity was developed in the routine manner. Because of the patient's age, extensive dissection was not attempted and only a modified radical mastoidectomy was performed. A T-shaped plastic flap was made in the external canal to line the mastoid cavity. The incision was closed and a compression dressing was applied. The patient withstood the procedure well and was returned to his room in good condition.

Postoperative Period:

Recovery was uneventful. The patient was up, and sitting by his bed on the first day. There was a large amount of sero-sanguineous discharge for several days. The dressings were changed daily; the drain was shortened every other day and finally removed completely on the ninth postoperative day. The sutures were removed on the sixth day and the postauricular incision was well healed. From the ninth to the 16th day the opening in the external canal was inspected and cleansed of debris and discharge by means of suction. On the 20th day the ear was almost completely dry, and examination revealed a clean, well healing cavity. The patient said that he felt improved and had not had an attack of vertigo or tinnitus since the operation. Roentgenograms of the mastoids 21 days after the operation showed that the large defect in the left mastoid had filled in considerably. A portion of the bone was missing posteriorly following surgery, but apparently the area of bone destruction was regenerating with new bone following removal of the foreign body.

A communication from the patient in November, 1953, approximately one year after the operation, related that he was asymptomatic and in good health despite his age.

DISCUSSION.

This case clinically suggested an expanding lesion of the mastoid antrum which was producing pressure necrosis. Roentgenologically it did not resemble a cholesteatoma. The cotton foreign body had been present in the mastoid antrum for 32 years and probably was the reason why the original operation did not give the expected result. This foreign body was firmly entrenched in the antrum and could have reached that point only through a previous operation. It would obviously have been impossible for the patient to accidentally push the cotton into this area while cleaning his ear.

The area of erosion in the antrum was larger than the cotton foreign body. It is doubtful whether the foreign body alone could have produced this result. The role of secondary

infection and granulation tissue formation is an important one. The fact that the patient lived for 32 years with only occasional difficulty indicates the slow process involved.

CONCLUSIONS.

1. This case presents an unusual aspect of chronic middle ear disease and illustrates the importance of careful inspection of the mastoid cavity and surrounding structures before completion of any type of mastoid surgery.

2. Radiologically the erosion in the mastoid antrum was not typical of cholesteatoma, neoplasm or mastoiditis, although it did resemble a tumor to some extent.

3. It is felt that the erosion was caused by a combination of the cotton foreign body, secondary infection and granulation tissue rather than by pressure of the cotton alone.

104 W. Madison Street.

ANNOUNCEMENT.

An introductory course in Reconstructive Surgery of the Septum and External Nasal Pyramid will be given, under the direction of Dr. Maurice Cottle, March 19 through 26, 1955, at the University of Oregon Medical School. This will be sponsored by the Department of Otolaryngology.

BOOK REVIEW.

Anatomy of the Bronchovascular System. Its Application to Surgery. By George L. Birnhaum, M.D., Chief, Surgical Unit, Veterans Administration, Regional Office, Portland. Foreword by Evarts A. Graham, M.D., Professor Emeritus of Surgery, Washington University, St. Louis. Cloth. Pp. 300, with illustrations. The Year Book Publishers, Inc., Chicago. 1954.

The practicability of planned intrathoracic surgical procedures has resulted in an increasing interest as well as an extensive literature in this important field. From this literature, the author has reviewed much of the new knowledge of the anatomy, embryology, congenital anomalies and surgical problems of the bronchovascular system.

The anatomical section contains an exhaustive, detailed analysis of the common bronchovascular variations not found in the standard text. Congenital heart disease is systematically discussed from the standpoint of the malformation and clinical findings but with emphasis on the surgical problem, solution and indications for surgical intervention. The experimental surgery of the pulmonary vessel, trachea and bronchi, the surgery of asthma and the technique of pulmonary resection is reviewed in the second half of the book. A series of line drawings illustrating unusual variations of pulmonary arteries and veins is to be found in the appendix.

The book is attractively bound and the paper and typography of excellent quality. For those interested in investigative work, the bibliography of over 500 references will serve as a helpful guide. Of interest primarily to the thoracic surgeon and bronchoesophagologist, this book should make instructive reading for the internist, radiologist and pediatrician as well.

P. H.

THE ASSOCIATION OF MILITARY SURGEONS OF THE UNITED STATES.

ANNOUNCEMENT.

The 61st Annual Convention of the Association of Military Surgeons of the United States will be held November 29 to December 1, 1954, at the Hotel Statler, Washington, D. C. The program will cover a wide range of professional and scientific subjects plus the latest advances in medical and allied sciences, the latter to be shown in a large variety of technical exhibits and films.

For the entertainment of the members and guests, a full schedule of events is being arranged. As in the past, one of the outstanding features is the Honors Night Dinner on December 1, at which the Sir Henry Wellcome medal and prize, the Gorgas medal, the Stitt award, the McLester award, the Louis Livingston Seaman prize, and the Founder's medal will be presented.

The granting of credit points for retirement to eligible Reserve Officers for attendance at meetings is expected to be announced soon by the Department of Defense.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC., MEETINGS.

MID-WINTER.

Eastern Section Meeting.....Friday, January 7, 1955
Philadelphia, Pa., Hotel Warwick

Triological Council Meeting.....Saturday, January 8, 1955
New York City, The Waldorf-Astoria

Western Section Meeting...Saturday-Sunday, Jan. 15-16, 1955
Los Angeles, Cal., The Town House

Southern Section Meeting.....Saturday, January 22, 1955
Charlottesville, Va., Medical School Auditorium

Middle Section Meeting.....Monday, January 24, 1955
Detroit, Mich., Sheraton-Cadillac Hotel

SPRING.

American Board of Otolaryngology
Richmond, Va., March 6-10, 1955

All Society Meetings will be held at the Hollywood Beach Hotel, Hollywood, Florida.

American Laryngological Association.....March 13, 14, 1955

Triological Society.....(mornings only) March 15, 16, 17, 1955

American Broncho-Esophagological Association
(afternoons only) March 15, 16, 1955

American Otological Society, Inc.....March 17, 18, 1955

The Hollywood Beach Hotel (American plan) is a delightful place to hold the Spring Meetings — you may wish to bring the family along. The Hotel offers its guests such features as ocean bathing, deep sea fishing, 18-hole golf course and country club, tennis and many other activities.

For hotel reservations please communicate with Mr. John W. Tyler, Manager, Hollywood Beach Hotel, Hollywood, Florida.

TWENTY-FOURTH ANNUAL MID-WINTER CLINICAL CONVENTION IN OPHTHALMOLOGY AND OTOLARYNGOLOGY, JANUARY 17 THROUGH JANUARY 28, 1955.

The Research Study Club of Los Angeles announces its Twenty-fourth Annual Mid-Winter Clinical Convention in Ophthalmology and Otolaryngology, January 17 through January 28, 1955. For the Ear-Nose-Throat, the main Guest Lecturers will include Dr. Theodore E. Walsh, Professor and Head of the Department of Otolaryngology, Washington University, St. Louis; and Dr. John J. Conley, Chief of the Head and Neck Service, St. Vincent's Hospital, New York City, and Clinical Professor of Otorhinolaryngology, New York University, and other appointments.

Dr. Walsh has long been known to us not only as a professor, but also as a lecturer in our national societies, with particular interest in otologic and rhinologic problems.

Dr. Conley has done a great deal of work in tumors and plastic surgery of the head and neck, as well as being a well trained man in the broader field of our specialty.

Also serving on our program with Didactic Lectures will be Dr. Kenneth C. Brandenburg of Long Beach, Dr. J. C. Almy Harding of San Diego, and Dr. Paul J. Moses of San Francisco.

Instruction Courses in Otolaryngology will be provided by Dr. E. R. V. Anderson, Dr. Norman Jesberg, Dr. Harold Owens, and Dr. Manuel R. Wexler, all of Los Angeles, and Dr. Leo Shahinian of San Francisco.

The main Guest Lecturers for the Eye program will be Dr. Edmund B. Spaeth and Dr. Irving H. Leopold, both from the University of Pennsylvania Graduate School of Ophthalmology.

The first week will be devoted to the Ear-Nose-Throat—from Monday to Saturday, January 17 through January 22. The Eye week will cover Monday to Friday, January 24 through January 28. Those of us who confine our work to only one of these specialties may complete either subject in one week.

On Tuesday evening, January 18, the Annual Banquet of the Los Angeles Society of Ophthalmology and Otolaryngology will honor the Guest Lecturers of that week. This will be held at 7:00 p.m. in the Ball Room of the Elks Club.

On Wednesday afternoon of each week motion pictures will be shown, Ear-Nose-Throat subjects the first week, and Eye subjects the second week. These afternoons will also afford time for relaxation, sight-seeing, golf, radio broadcasts, and the enjoyment of points of interest in and about Los Angeles, including the famous Huntington Library with its collection of rare books and art.

The American Laryngological, Rhinological and Otological Society, Western Section, will hold its Annual Meeting at The Town House immediately preceding this Convention, Saturday and Sunday, January 15 and 16. On Saturday afternoon the scientific session is open to members and all interested otolaryngologists. Saturday evening there will be a dinner meeting for members of the Triologic Society only. Sunday morning, the second scientific session is open to all interested. The president of the Society, Dr. Kenneth M. Day, after attending this Society's Annual Meeting, will be with us during a portion of the Mid-Winter Course of the Research Study Club.

Unless you have your own plans for a place to stay, it is advised that you write for reservations at an early date to Mr. H. M. Nickerson, Manager of the Elks Club, 607 South Parkview Street, Los Angeles 57. He will endeavor to arrange suitable quarters for you in a conveniently located hotel, and will confirm the reservation by return mail.

Each applicant must be a member, in good standing, of the American Medical Association *in Order to Become Eligible* for attendance at the Convention. The fee for the entire two weeks, or any part of it, is $100.00 and includes the cost of all luncheons. We are informed that these dues are an income-tax deductible item, as they represent an annual membership fee. Make your Check payable to "Mid-Winter Clinical Convention," and mail to Pierre Violé, M.D., Treasurer, 1930 Wilshire Boulevard, Los Angeles 57. If anything prevents your attendance, this fee will be returned to you.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

October 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 700.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model "Mercury."
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T-10; Microtone Model T-612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny Myte).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor; 2 tubes. Model A-330; 3 transistors. Model A-335; 3 transistors.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 35, New York.

Audivox, Model 71; 3 transistors.
Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Beltone Concerto Model; 3 transistors.
Manufacturer: Beltone Hearing Aid Co., 2900 W. 36th St., Chicago 32. Illinois.

Maico Transist-Ear, Model O; 3 transistors.
Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis 1, Minnesota.

Micronic "All American" Hearing Aid; 3 transistors.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T1 (Red Dot); 3 transistors. Microtone Model T1 (Yellow Dot); 3 transistors. Microtone Model T31 (Micro-Mite); 1 transistor and 2 tubes. Microtone Model Skylark; 3 transistors.
Manufacturer: The Microtone Corporation, Ford Parkway on the Mississippi, St. Paul 1, Minn.

Otarion Model C-15; 1 transistor, 2 tubes. Otarion Model D-1; 3 transistors. Otarion Model F-22; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Paravox Model K.M. (TRANSonic); 3 transistors.

Manufacturer: Paravox, Inc., 2056 East 54th St., Cleveland, Ohio.

Radioear Model 820; 3 transistors.

Manufacturer: E. A. Myers & Sons, Inc., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Sonotone Model 1010; 1 transistor, 2 tubes. Model No. 1111; 3 transistors.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes. Telex Model 956; 3 transistors.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Unex Model TR-3D; 3 transistors.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex Hearing Aids (three types).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Vice-President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Illinois.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Hollywood Hotel, Hollywood, Fla., March 17 (afternoon), 18 (forenoon and afternoon), 19 (forenoon), 1955.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Henry B. Orton, Newark, N. J.
First Vice-President: Dr. Jas. H. Maxwell, Ann Arbor, Mich.
Second-Vice-President: Dr. Clyde A. Heatley, Rochester, N. Y.
Secretary: Harry P. Schenck, Philadelphia, Pa.
Treasurer: Fred W. Dixon, Cleveland, Ohio.
Librarian-Historian: Dr. Bernard J. McMahon, St. Louis, Mo.
Meeting: Hollywood Hotel, Hollywood, Fla., March 13-14, 1955.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
President-Elect. Dr. Dean M. Lierle, Iowa City, Iowa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Eastern Section Meeting, Friday, January 7, 1955, Philadelphia, Pa., Hotel Warwick.
Triological Council Meeting, Saturday, January 8, 1955, New York City, The Waldorf-Astoria.
Western Section Meeting, Saturday - Sunday, January 15 - 16, 1955, Los Angeles, Cal., The Town House.
Southern Section Meeting, Saturday, January 22, 1955, Charlottesville, Va., Medical School Auditorium.
Middle Section Meeting, Monday, January 24, 1955, Detroit, Mich., Sheraton-Cadillac Hotel.
Meeting: Hollywood Hotel, Hollywood, Fla., March 15-16-17, 1955, morning only.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. F. W. Davison, Danville, Pa.
Vice-Chairman: Dr. Guy L. Boyden, Portland, Ore.
Secretary: Dr. Hugh A. Kuhn, Hammond, Ind.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October 9-15, 1955.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Hotel Marshall, Richmond, Va., March 6-10, 1955.
Palmer House, Chicago, Ill., October, 1955.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Clyde A. Heatley, 11 No. Goodman St., Rochester, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Hollywood Beach Hotel, Hollywood, Fla., March 15-16, 1955, afternoon only.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. John Louzan.
Vice-Chairman: Dr. Willard B. Walters.
Secretary: Dr. Jack L. Levine.
Treasurer: Dr. Russell S. Page, Jr.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Meeting: General Oglethorpe Hotel, Savannah, Ga., March 11-12, 1955.

THE GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. George McKenzie, Dupont Medical Bldg., Miami, Fla.
Vice-President: Dr. Maurice Edelman, 605 Lincoln Road, Miami Beach, Florida.
Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami, Fla.
Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. Norman Leshin, 55 East Washington St., Chicago, Ill.
Vice-President: Dr. Henry B. Perlman, 950 East 59th St., Chicago, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracuse, N. Y.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 9, 1955.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. F. Lambert McGannon, 14900 Detroit Ave., Lakewood 9, Ohio.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1955.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
Time and Place: 1956, Puerto Rico.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting:

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. S. Fitz-Hugh, Charlottesville, Va.
President-Elect: Dr. H. L. Mitchell, Lexington, Va.
Vice-President: Dr. Marion K. Humphries, Charlottesville, Va.
Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.

Spring Meeting: Natural Bridge Hotel, Natural Bridge, Va., May 7-8, 1955.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Harold Owens, M.D.
Secretary-Treasurer: Robert A. Norene, M.D.

Chairman of Section on Ophthalmology: Sol Rome, M.D.
Secretary of Section on Ophthalmology: Wendell C. Irvine, M.D.

Chairman of Section on Otolaryngology: Max E. Pohlman, M.D.
Secretary of Section on Otolaryngology: Herschel H. Burston, M.D.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles 57, Calif.
Time: 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section. 6:00 P.M., fourth Monday of each month from September to June inclusive—Otolaryngology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Harry Nievert, 555 Park Ave., New York (21), N. Y.
Secretary: Dr. Louis Joel Fleit, 66 Park Ave., New York (16), N. Y.
Meeting:

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Cecil Swann, Asheville, N. C.
Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. David S. Asbill, Columbia, S. Car.
Vice-President: Dr. John McLean, Greenville, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: Honolulu, 1954.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.
Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. William J. Hitschler.
Vice-President: Dr. Chevalier L. Jackson.
Treasurer: Dr. John J. O'Keefe.
Secretary: Dr. Joseph P. Atkins.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Thomas F. Furlong, Jr., Dr. William A. Lell, Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Oscar Marchman, Jr., Dallas, Texas.
Secretary-Treasurer: Dr. Morris F. Waldman, Dallas, Texas.

BALTIMORE NOSE AND THROAT SOCIETY

Chairman: Dr. Albert Steiner, 1308 Eutaw Pl., Baltimore, Md.
Secretary-Treasurer: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

CANADIAN OTOLARYNGOLOGICAL SOCIETY
SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. G. Edward Tremble, 1390 Sherbrooke St., West, Montreal, P. Q.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Meeting: Bigwin Inn, Lake of Bays, Muskoka, Ontario. June 16 to June 18, 1955.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretario del Exterior: Dr. Juan Manuel Tato.
Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asís R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
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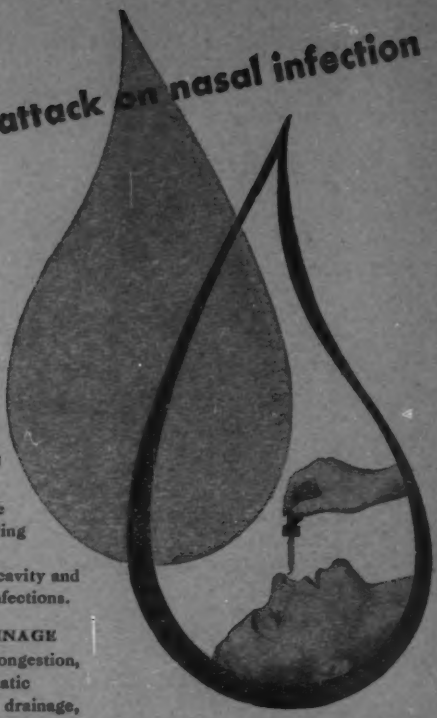

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